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U. S. Bureau of animal industry

U. S. DEPARTMENT OF AGRICULTURE.

FIRST ANNUAL REPORT

OF THE

BUREAU OF ANIMAL INDUSTRY

FOR

THE YEAR 1884.

WASHINGTON:
GOVERNMENT PRINTING OFFICE.
1885.

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Resolved by the Senate and House of Representatives of the United States of America in Congress assembled, That there be printed fifty thousand copies of the first annual report of the Bureau of Animal Industry of the Department of Agriculture, of which ten thousand shall be for the use of members of the Senate, thirty-five thousand for the use of members of the House of Representatives, and five thousand for the use of the Commissioner of Agriculture; the illustrations to be executed under the supervision of the Public Printer, in accordance with directions of the Joint Committee on Printing, the work to be subject to the approval of the Commissioner of Agriculture.

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LETTER OF TRANSMITTAL.

SIR: I have the honor to submit herewith the First Annual Report of the Bureau of Animal Industry, containing the investigations of the past year in regard to the contagious diseases of domesticated animals, and in regard to the present condition of the cattle industry.

The investigations in regard to the nature of the lung disease which prevails among the cattle of certain portions of some of the Eastern States, the contagiousness of which was doubted by many, have been very thorough, and demonstrate the communicability of the affection, and that its characters are identical with the epizootic contagious pleuro-pneumonia of Europe. The exposure of more than fifty healthy animals in four different lots to other cattle affected with this disease, and the communication of it to thirty of these, would seem to be sufficient evidence in itself to forever settle the question of contagion which has been so long debated.

The inspection of herds in the district which was supposed to be infected with this disease has been going on as rapidly as possible with the limited number of Inspectors allowed by the law for this work. When it is considered that each animal must be carefully examined by auscultating and percussing the lungs, it is really surprising that so much has been accomplished within the very limited period of about six months.

The outbreak of pleuro-pneumonia in the Western States was so serious a matter that it was thoroughly investigated by the Chief of the Bureau in person, and everything that could be accomplished under the law has been done to prevent its further extension. This outbreak, in which the introduction of the disease into every infected herd could be traced to contagion from a common starting-point, is also evidence in favor of the contagiousness of the disease which cannot be overlooked.

Considerable space has been devoted to the history of this plague in Europe, the means by which it was disseminated there, and the measures which have been adopted in some of the more important nations for its extermination.

The report also contains a carefully prepared account of the enzootics of ergotism which occurred in a number of the Western States during the winter and spring of 1884, and which attracted so much attention. A history of the disease is given at length, which shows that even in this country a number of similar outbreaks are on record in various States of the East, and that these have uniformly been considered to be the result of eating ergot with the food. A sufficient time has already elapsed to show that the predictions of the writer in regard to the non-contagious nature of the disease in Kansas were fully justified; it has never extended itself to other farms, and it seems to have entirely disappeared.

The investigations in regard to the nature of swine plague and the organism which causes it have been continued. The contest going on in Europe as to which of the several organisms described is the true parasite of the disease made it advisable for us to go over this part of the work again, and these later investigations have confirmed the conclusions which have been given by the writer in his former reports. It was necessary to settle this point before reliable researches could be made with a view of attenuating the virus and producing a vaccine for the disease.

The effort to locate the district permanently affected with Southern cattle fever has been successful with that part of the country lying east of the Mississippi River. The maps accompanying this report show very clearly the large extent of territory which it has been necessary to examine.

In addition to the veterinary investigations referred to above, there is a statement of the number of imported animals which have passed through each of the quarantine stations for the six months ending January 1, 1885. There are reports on different branches of the cattle industry by the Hon. J. B. Grinnell, by J. H. Fullinwider, and E. W. Perry. There is an important paper by Dr. E. M. Hunt, Secretary of the State Board of Health of New Jersey, on the history of pleuro-pneumonia in that State; and a very interesting account of the proceedings of the Fourth International Veterinary Congress which met at Brussels in September, 1883. Other reports of interest are also included.

Respectfully submitted, January 20, 1885.

D. E. SALMON,
Chief of the Bureau of Animal Industry.

HON. GEO. B. LORING,
Commissioner of Agriculture.

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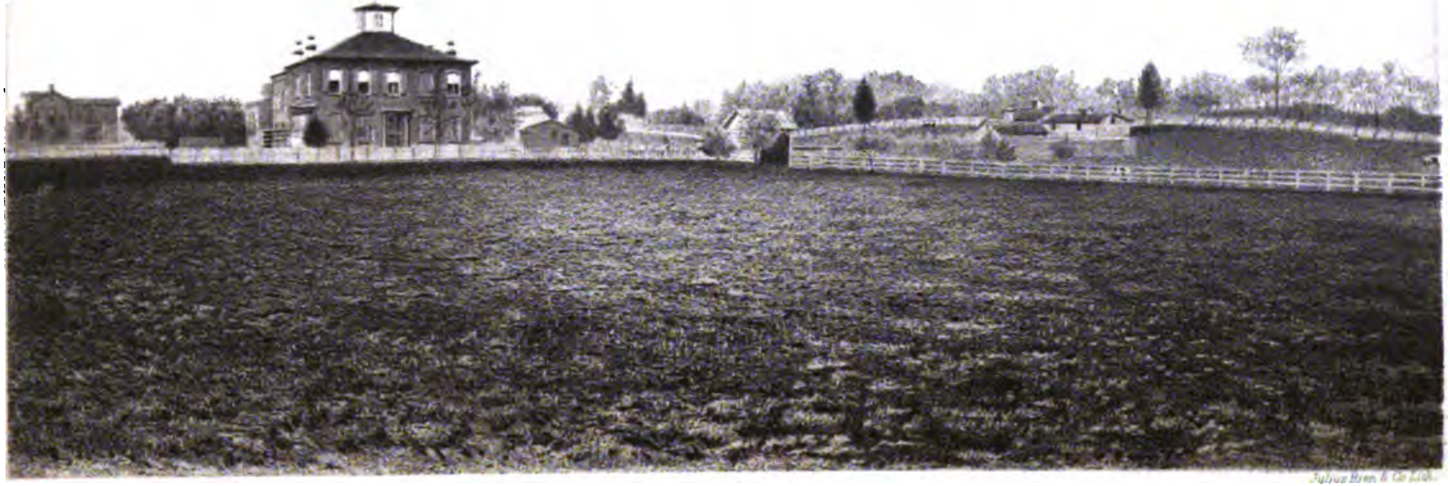

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VETERINARY STATION, DEPARTMENT OF AGRICULTURE, WASHINGTON, D C.

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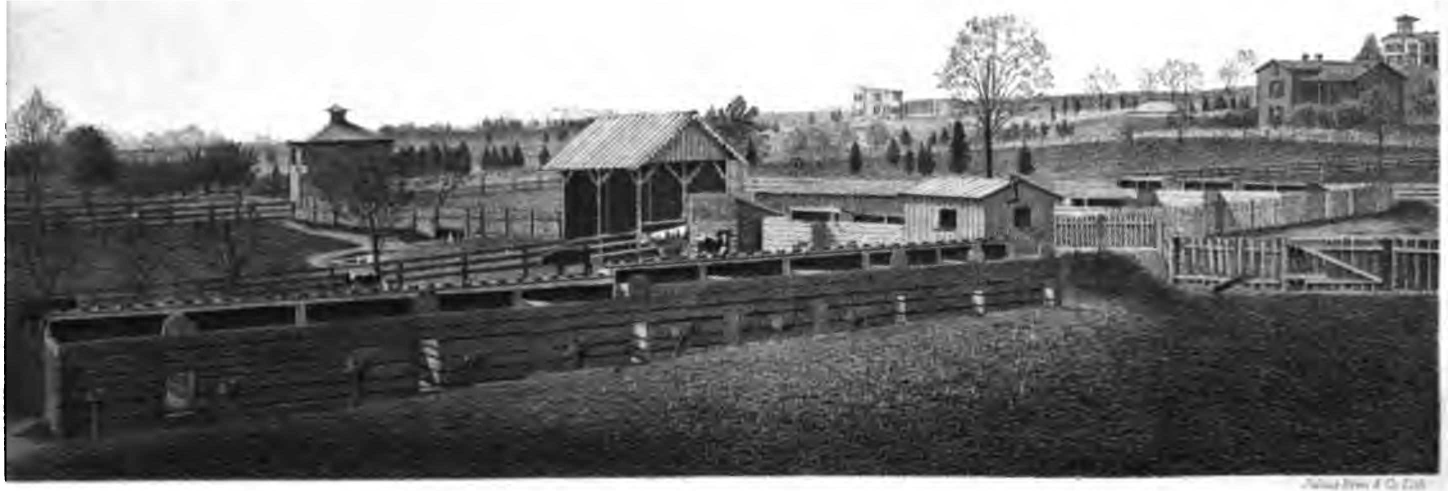

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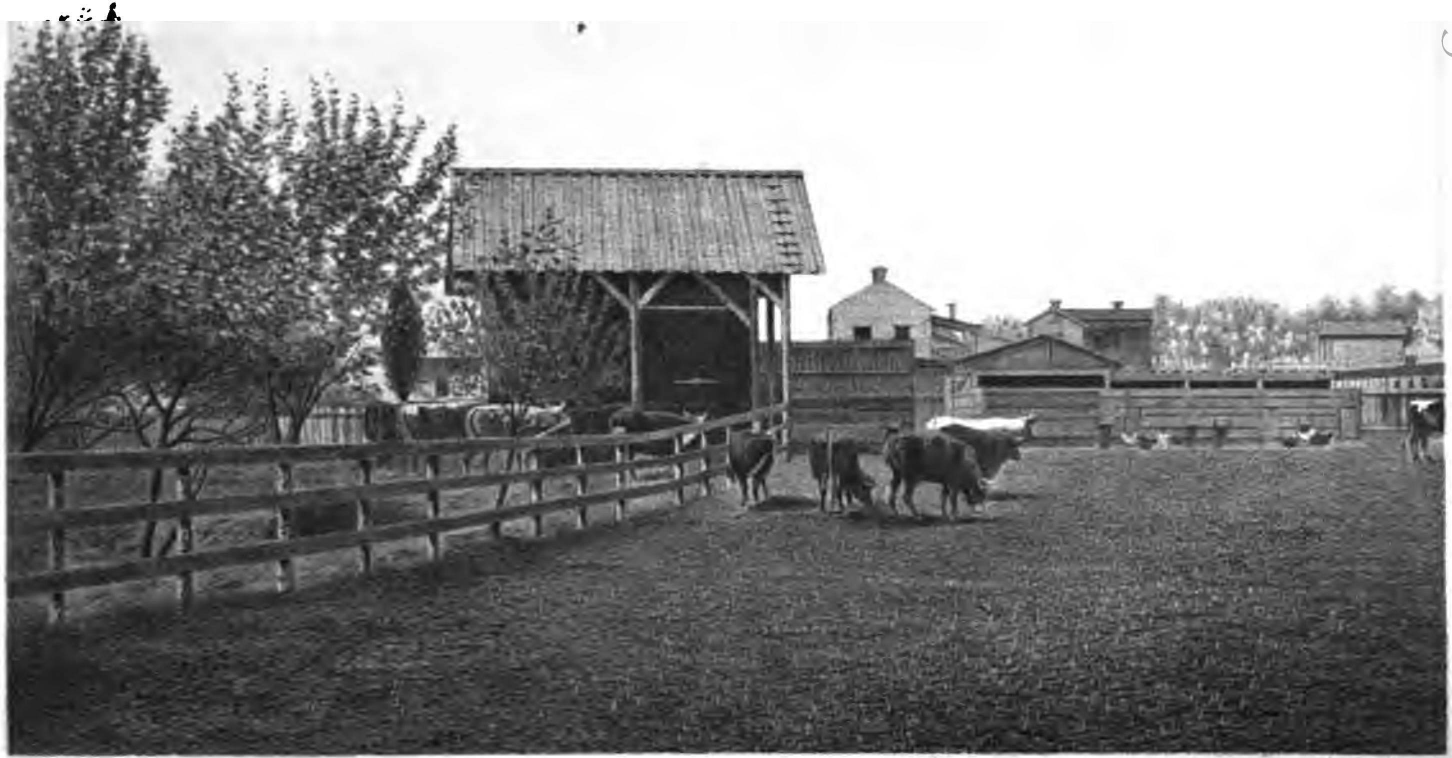
Plate III

Plate III



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VETERINARY STATION, DEPARTMENT OF AGRICULTURE, WASHINGTON, D. C

FIRST ANNUAL REPORT OF THE BUREAU OF ANIMAL INDUSTRY.

REPORT OF THE CHIEF OF THE BUREAU.

SIR: In my last report brief mention was made of the Veterinary Experimental Station established by your direction near this city. Since then many additions have been made to this Station, and it is now thought a more detailed description will prove of interest to those engaged in investigations as to the cause, transmission, and prevention of infectious and contagious diseases incident to domestic animals both in this and in other countries.

The Station is located on the Benning's Bridge road, about one-fourth of a mile east of the northeastern boundary of the city. The plat of ground on which it is located consists of 7 acres of rolling land, which is subdivided by new fencing into three pasture fields. The largest inclosure contains 4 acres, the second 2 acres, and the third 1 acre. There are seven outbuildings for the accommodation of cattle and the protection of the necessary implements for keeping the place in proper condition. Two wooden and two brick structures are used for the accommodation of cattle alone. The interior of the stables are fitted up with box stalls located on each side of a 4-foot passage-way extending the entire length of the buildings. The average measurement of each stall is 8 by 10 feet. One stable contains eight stalls, a second five, and a third four. When necessary, two steers or cows can occupy each stall with comfort. Ample feed-rooms are attached to each stable.

The fourth building is a wooden structure 25 feet wide by 31 feet in length, with interior free of compartments. This building is used for the protection from inclement weather of the cattle, which are allowed to graze on the largest pasture field, and has been left open on the south side.

Four rows of pig-pens are located at different points on the premises. One of these pens is 60 feet in length by 10 feet in width, and is subdivided into ten compartments. Two others are 30 feet in length, and contain five pens each. The fourth is divided into four pens. Ten or twelve pigs can be accommodated with comfort in each pen. They are supplied with cast-iron water-troughs, and the floors of each, as well as those of the stables, are laid in concrete, which prevents the absorption of water and facilitates disinfection.

In addition to the pens and stables a large chicken-house, 25 feet in length by 12 feet in width, has been provided for experiments with contagious diseases incident to fowls. A small building, midway between the pens and stables, has been fitted up for *post-mortem* examinations, and is supplied with all the necessary instruments for making autopsies.

A brick dwelling house, about 40 feet square and two stories in height, is located near the northern extremity of the grounds, and is occupied by W. H. Rose, V. S., superintendent of the Station. The water for the Station is supplied by two excellent wells conveniently located. Plates I, II, and III, accompanying this report, give accurate views of the buildings and grounds from different points.

INVESTIGATIONS OF PLEURO-PNEUMONIA.

DISTRICT OF COLUMBIA.

Since the recent publication of the report on "Diseases of Domesticated Animals for the years 1883–'84," a systematic inspection of cattle in the District of Columbia has been made, in the course of which a number of affected animals were discovered. By reference to the report of Dr. W. H. Rose, who made the inspection, the names of the parties owning these infected animals and herds, and the locality in which they may be found, are given. Preceding this inspection the disease had been found in ten stables in which more than one cow was kept, and in three others in which the diseased animal was the only one owned. In one stable two had died; in a second, one had died and two were sick; in a third five had died and six were more or less affected; in a fourth, two had been lost; in a fifth, six had been lost; in a sixth, five had died; in a seventh, three had died; and in the remaining three stables the loss, so far as we are aware, has been one animal each.

The total number of animals referred to above is twenty-seven which have died, and eight which were sick at inspection. In these cases the symptoms and *post-mortem* appearances of the animals examined were those of contagious pleuro-pneumonia, and the history, when it could be obtained, also pointed in this direction. The following instance is an illustration of this:

A cow belonging to Mrs. Flanigan, of Benning's road, was discovered sick, May 22, 1883. The symptoms were a severe, dry cough, emaciation, arched back, extended head, and turning out of the elbows. Percussion and auscultation showed that there was dullness and loss of respiratory murmur over the right lung.

This animal was preserved until August 27, and then slaughtered. The anterior portion of the right lung was found to contain a large encysted mass of hepatized lung tissue, fully 5 inches in diameter, which was beginning to disintegrate and break down into pus. The left lung was affected with chronic bronchitis, and many of the bronchi were filled with a thick, white, tenacious pus.

The disease was brought to this stable in the latter part of December, 1882, by a cow dealer who lives near the navy-yard. She presented symptoms of disease in about two weeks after purchase and lingered for six weeks with symptoms of acute lung disease. Three weeks after the death of this first cow a second became sick, with similar symptoms, and died after four weeks' illness.

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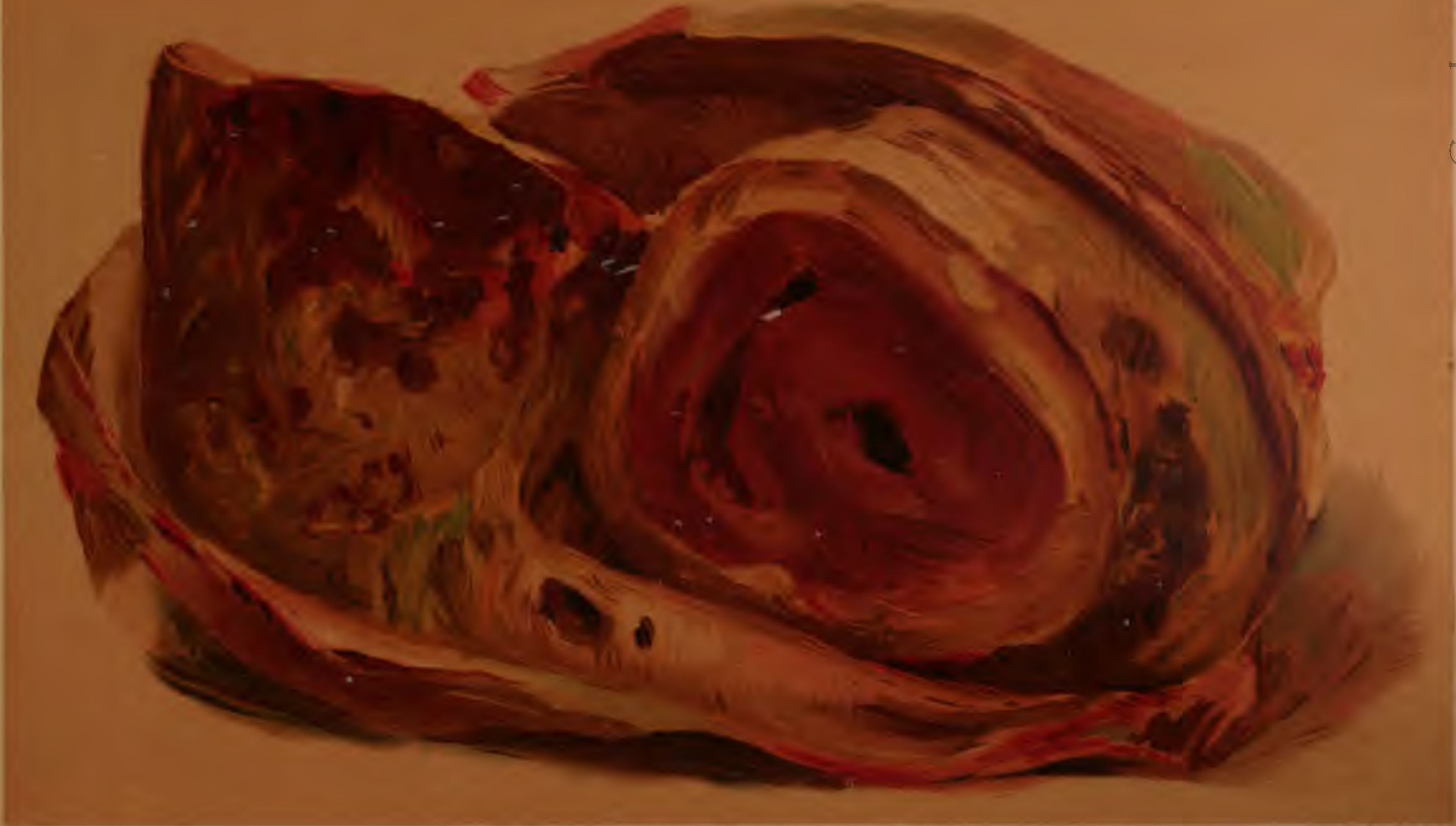

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ACUTE PERICARDITIS (COW)

UNIV. OF MICH.

Two others were successively affected in a similar manner and died; and, finally, the fifth came down with the disease about the 1st of May, 1883.

On May 29, 1883, we received at the Veterinary Experiment Station a cow from the stable of Catharine Bresnahan, of Lincoln avenue. This animal was somewhat tympanitic and stood with arched back, elbows turned out, and extended head. With each expiration there was a loud moan. Examination over the lungs revealed dullness, tenderness, and loss of respiration on the right side.

This animal died during the night of June 3, and was examined the following day. The right lung was found to be firmly attached to the ribs and diaphragm over nearly the whole surface of contact. This lung was almost completely hepatized; the posterior part was gangrenous; the median portion showed old hepatization, in which there was little difference in color between the lobular and the interlobular tissue, while the anterior portion was freshly hepatized and presented the distinctly marbled appearance seen in acute pleuro-pneumonia, and thought by some to be characteristic of that disease. The condition of this lung showed beyond question that the inflammation was a progressive one, and, beginning in the posterior portion of the organ, had successively invaded the median and anterior portions.

The existence of inflammation of different ages, showing the progressive character of the disease, is now regarded by the leading authorities of Europe as the most satisfactory means of distinguishing between contagious pleuro-pneumonia and the sporadic inflammations of the respiratory organs. The pleural cavity contained about a quart of effusion, and the mucous membrane of the bronchial tubes was of a deep red color.

This animal presented, consequently, all the symptoms and *post mortem* appearances described as peculiar to pleuro-pneumonia. The only history that could be obtained was that a number of cows had previously been affected in this stable with similar symptoms.

September 18, 1883, I examined a cow on Nineteenth street, which had rapid and difficult breathing, with extended head and elbows turned out as in cases of pleuro-pneumonia. There was dullness over the lower half of both lungs, with resonance above, but no respiratory murmur could be detected over the left side from the shoulder backward. This animal died on the morning of September 21, and on examination the left lung was found solidly attached to the ribs and diaphragm. There was an abundant effusion of liquid into the pleural cavity; the pericardium was greatly distended and attached to the costal pleura. On section the lung was found free from hepatization, but the pericardium was greatly thickened and transformed into a fibrous cyst inclosing the heart. The surface of the heart showed that this organ had been intensely inflamed; it was roughened and covered with granulations, mostly gray in color, but over parts of the surface mottled with deep red. The heart tissue, to a depth of half an inch from the surface, had undergone fibrous degeneration, was colorless, and resisted the knife. A painting was made of this organ and is reproduced in this report as Plate IV; it shows very plainly the thickened pericardium, the mottled appearance of the surface of the heart, which organ was cut across to reveal the depth of the fibrous degeneration.

There may be a question as to the exact nature of this disease—whether it was induced by the virus of lung plague or whether by other causes. No diseased animals had been introduced on the place, but there had been opportunity of exposure to animals running at large.

The absence of hepatization is not conclusive evidence that it was not lung plague. This disease quite often confines itself to the serous membranes without appreciably affecting the lung tissue, and pericarditis and epicarditis are manifestations which have been described as occurring in the infected stables of Europe. I am inclined to think, therefore, that this affection was the result of exposure to the lung-plague virus.

January 12, 1884, three cows were slaughtered at the Veterinary Experiment Station in presence of Hon. James Wilson, of Iowa, member of the House Committee on Agriculture, and of delegates from the Chicago convention of stockmen and of distinguished veterinarians, in order to demonstrate the character of the disease from which the cattle in this vicinity were suffering. The first one was a young cow that I found January 1, 1884, at the stable of the owner near Washington. At that time her breathing was rapid and labored, a distinct grunt or moan being emitted at each expiration. On percussion over the region occupied by the lungs the right side was found perfectly dull and without resonance, while the left side was resonant over the upper half, but very dull below. Auscultation showed complete loss of respiratory murmur over the whole of the right and over the lower part of the left side. There was no cough.

This cow had been purchased about a month previously, from a dealer who had brought her from the Shenandoah Valley, in Virginia, and had kept her for a number of days (the exact time not known) at his stable in Washington. She was noticed to isolate herself from the remainder of the herd while at pasture, and to be disinclined to move, almost as soon as she was placed with the herd. She commenced moaning at each expiration more than two weeks before I saw her, and was then separated from the other animals. January 2 she was removed to the Experiment Station, her temperature at that time being about 103° F.

This cow died during the night of January 11, and was examined about 11 o'clock the following day. On opening the thorax about 2 gallons of amber-colored liquid escaped. The right lung was solidly attached to the costal pleura and diaphragm by thick false membranes of recent formation. On the left side the attachments were not so extensive, and the membranes were of still more recent growth. On each side there were thick masses of coagulated lymph, weighing from 2 to 3 pounds, and of a whitish color and firm consistency, which indicated their formation a number of days before the death of the animal. The lung tissue presented no signs of hepatization.

The second cow examined was brought to the Station over two months before, and at the time of this examination was somewhat emaciated. She was coughing when first seen, had little appetite, and an examination of the lungs showed dullness and loss of respiratory murmur over the lower part of the right lung.

Three animals had previously been lost in the stable from which she came, and before death they presented symptoms of lung disease.

This cow was slaughtered, and on opening the cavity of the thorax the left lung was found adherent to the diaphragm and the right lung to the costal pleura. The right lung contained four or five masses, varying from two to four inches in diameter, surrounded by a thin cyst wall and composed of hepatized lung tissue in a disintegrating condition.

The third animal, which was also somewhat emaciated, was obtained January 10, from a stable where two cows had been lost in the preceding summer. She had been purchased for $50 two or three months before she sickened, and was at that time in good health. When brought to the Station her temperature was 104° F., and there was complete dullness and loss of respiration over the left lung. Her condition was substantially the same on the day of examination, January 12. When, after slaughter, the ribs of the left side were removed, a considerable quantity of amber-colored liquid escaped. This lung was completely hepatized and solidly attached to both the ribs and diaphragm. A section of the lung disclosed the interlobular tissue distended with lymph, though not to the degree sometimes seen. There was, however, a very distinct marbled appearance, and a difference of coloration between the upper and lower parts of the lung that probably resulted from a difference in the age of the hepatization in these two portions. The right lung of this animal was in a normal condition.

A fourth cow was obtained from a Washington stable the same day that the above examinations were made. She died during the night of January 12. Her appearance before death and the condition of her lungs when examined were very similar to that of the third cow mentioned above.

May 1, 1884, a sick cow was reported at Miss Fannin's, on M street, in this city. She was examined the same day and found to be moaning with each expiration; her breathing was labored; there was salivation, extended head, and elbows turned out. The bronchial breathing was loudest on the right side; the left side was very dull on percussion up to and somewhat above the median line. The right side had a dull area at lower portion of thorax and another above the median line.

May 5, this animal, now sinking rapidly and already tympanitic, was slaughtered. The autopsy revealed the left lung completely solidified with the exception of a very small part of the anterior lobe. Various stages of inflammation were to be seen in the different parts of the lung. There were thick false membranes and solid adhesions to the diaphragm and costal pleura. The right lung was extremely emphysematous, and parts of it adherent to the costal pleura, but there was no hepatization of its tissues.

CONNECTICUT.

In the latter part of August, 1883, I investigated an outbreak of disease at Salem, Conn., which had affected cattle on the farms of H. E. Williams and Captain Seaman, of that place. The history of this outbreak may be summarized as follows: Hon. E. H. Hyde, of the State Commission on Diseases of Domestic Animals, first visited the farm of Mr. Williams on August 8, and at that time found a young bull in the lot partially recovered from an attack of disease, and a cow and an ox were both very sick with what he considered to be the typical symptoms of pleuro pneumonia. At Captain Seaman's a cow was very sick and presented the same symptoms as were seen with the affected cattle belonging to Williams.

The next morning Dr. Rice, of Hartford, was called, and on arrival, Williams' cow was found to have died during the night.

A *post-mortem* examination was made and the lung found attached to the walls of the chest; when cut across it was seen to be solidly hepatized, of a marbled appearance, and presented all the characters of contagious pleuro-pneumonia. The Commission advised slaughter, which was objected to, but the same day, after the departure of the State officers, the sick ox belonging to Williams and the cow belonging to Seaman were slaughtered. These animals were not examined professionally, but the descriptions which I received from those who were present were sufficient to satisfy me that the lungs were solidified and attached to the ribs.

August 29, I visited Mr. Williams' farm and learned from him the particulars of the outbreak. The first symptoms of disease were seen in one of the cows June 20, and a second cow was attacked on June 23; both of these died from the effects of the disease July 3. At the time of my visit, August 29, there were six animals on the place; one ox, quite sick, with left lung solidified; one Jersey cow, had been quite sick but was now better; one young Jersey bull, with left lung solidified, and three Jersey cows, in which I found no evidence of disease. Only one animal had been brought on the place within a year preceding the outbreak, and that was a Jersey cow named Mollie Lathrop 3d, No. 7627. She was obtained by exchange with Charles Decline, of New Durham, N. J., on April 10, 1883. This cow aborted the last of May, but has shown no other signs of sickness. At the time of examination she was in fine condition, fat, glossy, with no cough and no signs of lung disease revealed by either auscultation or percussion.

I visited Charles Decline at New Durham, N. J., on August 30. He stated to me that he exchanged cows with Williams about April 16. His cow went to New London on the same boat that the other returned by. According to the statement of Williams' farmer, the two cows were together about a quarter of an hour at New London. The cow Decline received from Williams sickened about the last of May. About a week later she and another Jersey cow, which stood beside her, and which was also sick, were killed and examined by his son, who is a veterinary surgeon. Both were affected with lung disease, which he pronounced to be pleuro-pneumonia. The lungs were hepatized, marbled in color, and attached to the walls of the chest.

Decline purchased Mollie 3d of Mr. Whitenack, of Dunellen, N. J., December 13, 1881. He says that he never had any disease among his cattle until after the cow arrived from Connecticut, and attributes the infection to her.

It was evident that some of the facts connected with the history of the disease in these two herds had been concealed, but it was very certain that the disease had existed in both herds, and it was very probable that one of the herds had been infected as the result of the exchange referred to above. Considering that there had been no disease in Connecticut until nine weeks after the exchange, and that it was admitted to have existed in Decline's herd four weeks earlier than it appeared among Williams' cattle; and considering, further, that the vicinity of New Durham has long been infected with pleuro-pneumonia while none had previously existed in the neighborhood of Salem, and the probability is that the disease was carried from New Jersey to Connecticut. There is one other possibility, however, viz., that both cows were infected on the boat or between the boat landing and Decline's place.

This theory is not probable, for the reason that a second cow was sick at Decline's by the last of May, and this would require the assumption that two full periods of incubation had elapsed between April 16 and May 30; that is, within six weeks. Now, it is very seldom that the period of incubation of pleuro-pneumonia is less than four weeks, and it is generally longer than this; consequently, it is very unlikely that in two successive cases on the same farm it would be reduced to three weeks. The admitted fact that both sickened at about the same time is an indication that both were infected at the same time, and from a common source, rather than that one contracted the disease from the other.

A second visit was made to the farm September 7, in company with Hon. E. H. Hyde and T. S. Gold, of the State Commission on Diseases of Animals, and Doctors Thayer, Rice, and Parkinson. At this time the bull and ox still presented symptoms of pleuro-pneumonia. The cow, Mollie 3d, was again carefully examined and showed a rather large area of dullness over the region of the heart and another low down on the right side. My own opinion was that this dullness did not indicate any disease of the lungs, though some of the others thought differently. It was admitted by all, however, that there were no positive signs of diseased lungs in her case.

A third visit was made, in company with the same gentlemen, with the exception of Dr. Thayer, September 12, when the ox mentioned above was slaughtered and examined. This animal was now believed by the owner to have recovered. The autopsy revealed the left lung solidly attached over a large surface to the thoracic wall and diaphragm. One-third of the organ was encysted and beginning to disintegrate, another third showed more recent hepatization and was not yet encysted. A section showed the characteristic marbled appearance, and the difference in the age of the inflammatory process in various parts of the lung.

Members of the State Commission have since informed me that the bull continued to fail and was destroyed by the owner on the 27th of October. Before this, however, the Commission was called September 18 to see a new case of the disease, which had developed on the farm of Amos Williams, the second neighbor south from the originally infected premises. This was a cow, which presented the typical symptoms and *post-mortem* appearances of pleuro-pneumonia, having been condemned and killed by the Commission.

To recapitulate: H. E. Williams had seven animals affected out of his herd of nine by the introduction of the cow from New Jersey, which animal was so slightly diseased as never to attract attention. Of the seven sick ones three died of the disease. Two of those slaughtered probably could not have recovered; one of the slaughtered oxen was improving, while the remaining cow was very sick when I last saw her. The adjoining farm on the north and the second one on the south each lost one animal from the disease. There were, consequently, nine animals affected in this outbreak.

PENNSYLVANIA.

October 3 and 4, I visited Chester County, Pennsylvania, in company with Mr. T. J. Edge, special agent of the governor, and Dr. Bridge, State Veterinarian. On the farm of W. P. Thomas I witnessed the slaughter of 3 cows, and on the farm of J. H. Garret I saw 5 others killed, these having been condemned by the State authorities as affected with contagious pleuro-pneumonia. The autopsies revealed the existence of a very similar condition in each of the animals. In most cases a whole lung was hepatized and firmly attached to the diaphragm and ribs. In several of the animals both lungs were affected. The pleural cavity contained large quantities of straw-colored effusion, and the connective tissue of the lungs was excessively distended with exudation of a similar liquid. The inflammation was very plainly of a progressive character, and the marbling of the lung was as distinct as in any cases I have ever seen.

The disease was introduced into this section by a car-load of 14 cows brought by John Noble from Baltimore. Where these cows were originally infected is a contested point between the authorities of Pennsylvania and those of Maryland; but there is no reason to doubt that the outbreak near West Chester was caused by this lot of animals.

These cows were sold as follows: July 19, to W. H. Shepherd, 1; July 26, to W. P. Thomas, 3; July 26, to H. Euches, 4; July 27, to J. H. Garret, 2; August 1, to J. Kelly, 2; not traced at time of report, 2.

Mr. Shepherd's cow was found sick with symptoms of pleuro-pneumonia September 8, and slaughtered by the State authorities. The autopsy revealed the characteristic lesions of lung plague. September 13 a cow was found affected with the same disease and slaughtered on Mr. Garret's farm. September 29, it was necessary to slaughter one of Mr. Thomas' cows. October 1, it was found that two cows had already died on Mr. Euches' farm, and that six others were sick.

According to information received from Dr. Bridge, October 23, 1884, the number of cattle exposed and slaughtered on account of sickness was as follows:

Owner.	Number exposed.	Number killed.
W. P. Thomas	42	42
Homer Euches	29	12
J. H. Garret.	33	14
W. H. Shepherd	1	1
Total	105	69

Eight adjoining herds were infected by the above, as follows:

Owner.	Number exposed.	Number killed.
W. H. Pratt	17	6
M. S. Garrett	11	3
E. J. Lewis	15	5
C. Smedley	22	2
Geo. P. Hughes	20	8
W. F. Dutton	16	16
W. Evans	5	1
L. V. and W. E. Smedley	21	9
Total	127	50

The affected cows which I saw were native animals in good condition. They had excellent pastures to run on, and there was no local cause whatever which could be suspected of producing this or any other disease. Besides, the time of year was not one in which acute lung diseases are seen among cattle. Nearly every one of the affected lungs which I saw when in this State showed the typical lesions of pleuro-pneumonia so plainly that, according to the best authorities in the veterinary profession the world over, any one of them would have been sufficient to afford a safe basis for diagnosing the disease.

Besides the herds infected by the contagion introduced with the lot of cattle from Baltimore, six herds have been infected from other sources since September, 1883. The following table shows the number exposed in each of these and the number destroyed after showing symptoms of the disease:

Owner.	Number exposed.	Number killed.
F. Carr	8	1
W. Williamson	5	2
F. Galloy	20	14
—— Heisey	9	5
—— Myers	17	2
J. Noble	10	0
Total	64	24
Total in preceding tables	232	119
Total for State of Pennsylvania	296	143

NEW JERSEY.

Dr. Rowland, an Inspector of this Department, stationed at Jersey City, N. J., discovered during the summer of 1883 that animals affected with pleuro-pneumonia were being shipped to New York from Hunterdon County, New Jersey. An investigation was ordered by Dr. E. M. Hunt, secretary of the New Jersey State Board of Health, and a number of herds were found in Hunterdon County which had been for some time affected with this disease. Owing to the fact that the owner of the affected herds was a large cattle dealer who gathered up cheap animals from various parts of New Jersey and Pennsylvania, and to the additional fact that the disease had been upon his premises for an indefinite time, the origin of the trouble could not be satisfactorily traced.

The owners of the infected herds had resorted to inoculation to arrest the progress of the disease, and it was said that all fresh animals which arrived were speedily inoculated. In spite of this, however, the losses were very heavy, though their full extent could not be ascertained. Dr. Miller, who investigated the condition of these animals, November 1, informed me that out of one herd, containing 60 head, 22 had been lost; from another containing 65 head, 8 were known to have died, and 1 was killed to obtain virus for inoculation; from another, containing 46 head, 8 had died; from a fourth, containing 70 head, 10 had died; and from a fifth, 6 had died. There had, consequently, been at least 55 deaths; in addition, a certain number had partially recovered, and some diseased animals had been sold.

According to the best information we could obtain the total number of cases of pleuro-pneumonia which had occurred in this county was not less than 100. These herds were quarantined and the State authorities are doing everything possible with their limited appropriation to stamp out the disease; but where so many animals have been exposed, and where the contagion has been sown broadcast over the pastures of half a dozen farms, experience shows that it is next to impossible to remove all danger except by killing all animals exposed and quarantining the farms for a long time.

MARYLAND.

Owing to the variety of reports in regard to the existence of pleuro-pneumonia in Maryland, Dr. Rose was directed to proceed to Baltimore during the last week of October, 1883, and examine a sufficient number of stables to form a basis for conclusions in regard to the distribution of the disease in that section. The cases of sickness mentioned are only those in which the symptoms indicated pleuro pneumonia. The following is a list of stables in the order in which they were examined, with a condensed summary of the information obtained:

Stable No. 1: Contains thirty-five cows. One chronic case, two recent deaths.
Stable No. 2: Thirteen cows. No disease.
Stable No. 3: Sixteen cows. One chronic case, two recent deaths.
Stable No. 4: Seven cows. No information.
Stable No. 5: Nineteen cows. Admit that cows are exchanged as soon as they show signs of disease.
Stable No. 6: Nine cows. Three recent deaths.
Stable No. 7: Two cows. Admits recent deaths from lung disease.
Stable No. 8: Thirteen cows. Two recent deaths from acute lung disease.
Stable No. 9: Seventeen cows. Have lost many in the past. All are now well.
Stable No. 10: Eighteen cows. Have lost two during the summer.
Stable No. 11: Nineteen cows. Would neither allow an examination nor give information.
Stable No. 12: Seven cows. None sick. No information.
Stable No. 13: Eleven cows. None sick.
Stable No. 14: Fifty-six cows. One acute and four chronic cases of pleuro-pneumonia. Have lost heavily in past years.
Stable No. 15: Eighteen cows. Five sick with acute lung disease within two months, of which three died.
Stable No. 16: Forty-two cows. Acknowledge a loss of over 200 cows from lung disease within three years. Several now coughing.
Stable No. 17: Fifty animals. No disease.
Stable No. 18: Thirty-six animals. No disease.
Stable No. 19: Original herd 12 animals. Three died during September and October. Calf died in October which State Veterinarian examined and pronounced affected with pleuro-pneumonia. Three still sick with same disease. First cow to sicken came from another stable in Baltimore within a few weeks.

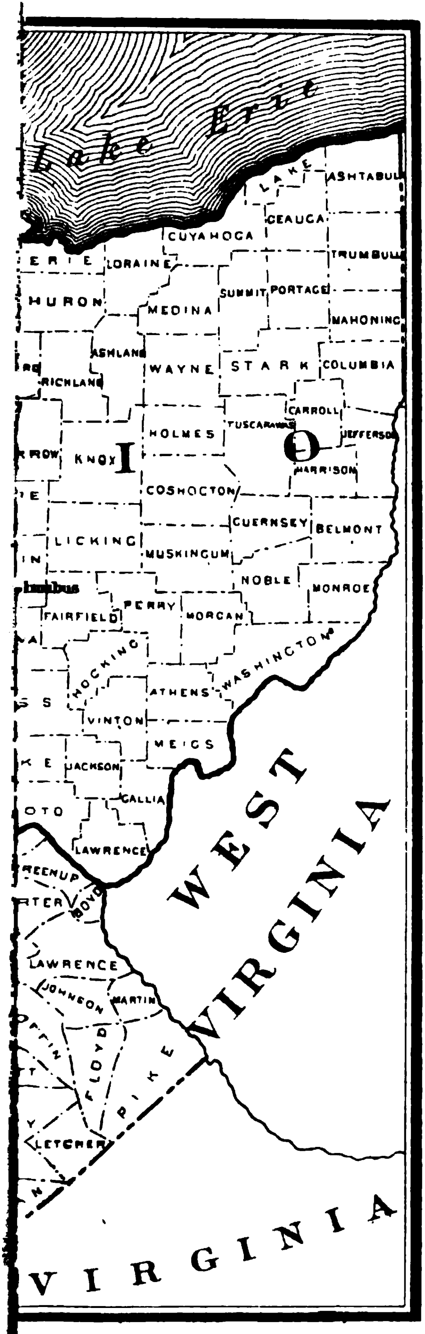
The herds in the nineteen stables referred to above contained 398 animals, of which 12 were found to be sick or only partially recovered at the time of inspection; 3 cows had recently been exchanged while sick, and 18 recent deaths had occurred. The total number of animals which had recently sickened with symptoms of pleuro-pneumonia in the above stables was, consequently, 33, or 8.3 per cent.

This inspection, while it cannot be taken as a very accurate indication of the proportion of the Baltimore dairy cattle which are constantly affected with pleuro-pneumonia, is nevertheless sufficient to show that a very large proportion of the stables are infected, and that many cases of the disease occur.

THE OUTBREAK OF PLEURO-PNEUMONIA IN THE WEST.

On the 15th day of July, 1884, Dr. Trumbower was requested to visit a cow at Sterling, Ill., belonging to Mr. O. A. Keefer. He found one of his thoroughbred Jersey cows, aged about six years, with the following symptoms:

The skin was abnormally dry and dead-looking, the animal standing with the head extended and the ears slightly drooping, coughing frequently and protruding the tongue. The character of the cough was dry, harsh, and rather weak, but not very painful. The eyes were bright and prominent, respiration 50, pulse 94, weak but regular, temperature 103.8° F. Auscultation on the right side of the chest revealed a subcrepitant sound immediately behind the shoulder, a little below the median line. In the middle and superior regions the respiratory


Lake Erie
LAKE
ASHTABUL
CEAUGA
CUYAHOGA
TRUMBUL
ERIE
LORAINE
SUMMIT
PORTAGE
HURON
MEDINA
MAHONING
ASHLAND
WAYNE
STARK
COLUMBIA
RICHLAND
CARROLL
TUSCARAWAS
HOLMES
JEFFERSO
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COSHOCTON
GUERNSEY
BELMONT
LICKING
MUSKINGUM
NOBLE
MONROE
FAIRFIELD
PERRY
MORGAN
HOCKING
WASHINGTON
ATHENS
VINTON
MEIGS
JACKSON
GALLIA
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JOHNSON
MARTIN
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murmur was slightly augmented; percussion elicited a trifling dullness over the lower third of the fourth and fifth ribs. In other parts no abnormal sound was produced, with the exception of a slightly increased resonance over the middle and superior regions. On the left side a loud murmur or sonorous rhonchus was heard in the median region behind the shoulder, accompanied by a dry and soft rubbing sound; below this part no respiratory murmur was audible, but in the act of coughing a gurgling or splashing sound was heard as that of a liquid being suddenly agitated in a cavity. Percussion revealed dullness over the central and lower posterior portions of the lung. No abnormal sensitiveness was manifested by pressure being applied along the spine or percussion over the chest. The history of this case is as follows:

Mr. Keefer saw this cow, Lass O'Lowrie, on the stock-farm of W. C. Clarke, Geneva, Ill., on the 6th day of April. At the same time he also saw there two other cows, Tama Warren and Nutrina of Tunlaw; all three had the appearance of unthriftiness, the hair looking rough and dry, but this was attributed to a severe winter without proper care, and, in the case of Lass O'Lowrie, to recent calving. On the 6th of June Mr. Keefer bought the latter animal from Mr. Clarke upon the representation that she was perfectly healthy. She was shipped on the 8th, and was four hours in transit. When Mr. Keefer took her from the car and drove her to his place she coughed frequently, and her hair looked bad. She was thin in flesh and yielded no milk. She calved some time in March and was again pregnant. From this time on she gradually became poorer and weaker. The milk secretion remained entirely suspended. She stood in the field away from the other cattle, and usually rested on the right side when in a recumbent position. Rumination was entirely suspended, appetite capricious, cough increasing in frequency, and had paroxysms of almost incessant coughing, lasting in the early morning for an hour or longer; nose alternately moist and dry; occasionally a string of mucus would be noticed to drop from the nostrils; the cough became more painful and the tongue was protruded in the act; frequent grating of the teeth was heard; no irregularity of the pulse or tympanitis was noticed; no arching of the back or turning out of the elbows; no moan or grunt accompanied respiration; no rusty colored and no discolored expectoration was coughed up. The case was thought to be one of tuberculosis, and isolation was recommended, and slaughter and burial as soon as he could decide upon the necessity of the measure. He was requested to give notice and allow a *post-mortem* examination to be made when she was slaughtered, or in case she should die. On the morning of the 24th she was bled to death. On examination, the anterior lobe of the right lung was found filled with tubercles covering a space 4 inches in diameter; they presented different stages of development, some containing a thick yellow or inspissated pus, while others were undergoing a caseous degeneration or calcification, and still others appeared as small, indurated, brown, or reddish, circumscribed spots in the interlobular tissue. There was very slight adhesion between the visceral and parietal pleura at the inferior and anterior extremity of the right lung; the superior and posterior four-fifths of the right lung was apparently healthy; on the left side there existed extensive adhesion of the posterior lobe of the left lung to the side of the chest and diaphragm, implicating almost the whole of the adjacent side of the pericardium; a large amount of firm gelatinous exudation and strong fibrous bands united the inferior, posterior, and central portions of the posterior lobe firmly to the pericardium, diaphragm, and costal pleura;

no abnormal effusion was present in either side of the chest; no indications of recent affection of the pleura were seen; the anterior lobe of the left lung contained numerous tubercles and abscesses from the smallest visible size to half an inch in diameter; many tuberculous nodules coalesced so as to form large irregularly-shaped masses; in the posterior lobe, beginning at the bifurcation of the trachea and extending downwards and backwards, was discovered a cavity 10 inches in length, which contained a pint of fluid of a grayish-black color of very offensive odor, holding in suspension disintegrated lung tissue; also in this cavity was discovered a mass of infarcted, necrosed lung tissue, weighing 2 pounds; the part nearest the right lung was breaking down and liquefying. Another mass of dead lung, weighing 4 ounces, of a yellow, granular, or caseous appearance, indicating that it was much older than the larger mass, was found lying in and partially buried in a separate sack which communicated with the larger cavity. The mass of infarcted necrosed lung on section presented a reddish-brown appearance, and the lobules were distinctly outlined as well as the remains of the larger blood-vessels and bronchi. The walls of the cavity were composed of fibrous tissue one-quarter inch thick, and remains of blood vessels extending into and across the cavity. The antero-superior portion of the right lobe of the liver presented one compact mass of tubercular nodules, and throughout the central parts of this organ numerous tubercles were seen. One measuring 2 inches in diameter was located at the transverse fissure, and was undergoing calcification. In the abdominal lymphatic glands there were masses of compact tuberculous matter encysted in strong fibrous capsules, one of which measured 3 inches in diameter. Nearly all of the tubercles presented a bright yellow color on section, and but few gray tubercles were seen, and then only in the lung tissue.

Dr. Trumbower sent parts of the lungs and liver of this animal to Washington for my examination. The cow had evidently been affected with tuberculosis, but the encysted mass of dead lung was a lesion which is not produced in this disease, but which is a frequent result of contagious pleuro-pneumonia. It seemed possible, therefore, that the two diseases might have existed at the same time in this animal, though the fact that we knew of no pleuro-pneumonia in that section of the country made the presence of this disease appear very doubtful.

On investigating the condition of affairs at Mr. Clarke's farm it was learned that his animals had been suffering from a disease that had caused the death of several during the spring and summer. A cow which had been sold to C. P. Coggeshall and taken to the farm of Mr. John Boyd, of Elmhurst, was very sick, and a second cow bought by Mr. Boyd was also sick. On the 12th of August I visited Mr. Boyd's place, and found that the cow called Cream Ecca, belonging to Mr. Coggeshall, had died the 20th of July; that the cow Edith St. Hilaire had improved very much during the past two weeks, and was then believed by her owner to be nearly well. Another cow, called Dessie 4th, belonging to Mr. Boyd's herd, was very sick. This animal was suffering from acute lung disease, with complete dullness and loss of respiration over the right lung, and dullness over the lower part of the left side of the thorax. Her temperature was 105° F. An examination of Edith St. Hilaire revealed dullness and loss of respiratory murmur over the posterior part of the right lung. An examination of Cream Ecca had been made by a local physician, who pronounced her affection to be consumption, and called the changes which he saw in the lung tissue caseous degeneration. A piece of this lung tissue, which was secured and shown to me by Dr. Trumbower, was hepatized as in pleuro-pneumonia.

These facts appeared sufficient to justify the diagnosis of contagious pleuro-pneumonia, but in the absence of any history beyond the Clarke herd, and considering the fact that the only cow of which a careful *post-mortem* examination had been made was certainly affected with tuberculosis, it seemed best to reserve a decision until more complete evidence had been obtained. In regard to Cream Ecca, it was said that her sickness came on soon after calving, which occurred July 2; that she retained the afterbirth and failed from that time. Two or three days later the afterbirth was removed by force and she commenced to sink rapidly and died July 20. The autopsy was not made until two days later. There was consequently some reason for thinking that her lung disease might have been the result of septic infection as a consequence of the forcible removal of the afterbirth. In regard to Dessie 4th, it was said that she had been tied by one of the farm hands to a wagon in the lot where she had been exposed to a cold rain and had undoubtedly suffered in consequence. It was believed by her owner that this exposure had aggravated, if it had not caused, her disease. In regard to Edith St. Hilaire, it was thought that she might have taken cold, and there were some indications that she had a tendency to tuberculosis. Taking all these facts into consideration the only safe course appeared to be to make further investigations before deciding as to the nature of the disease, and I urged Mr. Boyd to allow the slaughter of one or both of his sick animals; this he willingly consented to do, but it was now so late in the afternoon that we postponed the slaughter until the following day. On visiting the farm August 13, I found Mr. Boyd somewhat undecided as to whether he ought to allow the slaughter of his valuable animals for examination without further evidence that they were affected with a contagious disease. While discussing the matter one of the Chicago newspapers was received which contained a sensational article announcing the outbreak of contagious pleuro-pneumonia among Mr. Boyd's cattle, and a few minutes later two reporters appeared, who had been sent to make an investigation. It was now thought best to delay the slaughter and examination until the following morning, when this could be accomplished in quiet and without undue publicity. In the mean time arrangements had been made to isolate the sick animals from the remainder of the herd, and two cows which had shown loss of appetite and an increased temperature without any perceptible lesions of the lungs were also placed by themselves. On the morning of August 14, I made a third visit to Elmhurst, and in the presence of Mr. J. H. Sanders, member of the late Treasury Cattle Commission, Mr. Wadham, and Mr. Boyd, the two sick cows were slaughtered. An examination of the lungs of Edith St. Hilaire showed the posterior half of the right lung to be adherent to the ribs and diaphragm. The whole posterior part of the lung was inclosed in a fibrous cyst, the lung tissue being hepatized, of a reddish color, but still intact and firm. The median part of the left lung was in the same condition. With Dessie 4th the inferior and posterior parts of the right lung adhered by thick false membranes to the ribs and diaphragm. There was an abundant effusion of straw-colored liquid in both sides of the chest. Two-thirds of the right lung was hepatized, the thickened interlobular bands being distended with exudation liquid. There was plain evidence from the color and appearance of the different parts of the lung that the inflammation had been of a progressive nature, and that some lobules had been invaded much later than others. The left lung was covered at its posterior border with thick, white, false membranes; these were also seen over its anterior lobe; there were blood discolorations of the pleura and signs of hepatization in its earliest stage. In this case the intensity of the inflammation, the amount of lung tissue involved, the progressive nature of the inflammation, and the marbled appearance of the lung made it impossible to hesitate longer in concluding that the disease was contagious pleuro-pneumonia. It was in every respect a typical case of this disease.

Mr. Boyd had been informed at my first visit that the disease was probably pleuro pneumonia, and that he should at once take every precaution to prevent the infection of other animals, both in his own herd and in the herds of his neighbors; and he was informed as soon as the examination of two animals was concluded that there could no longer be doubt as to the nature of the disease, and that it would be necessary to take every possible precaution to prevent its spread.

August 15, I went to Geneva, Ill., and examined the condition of the animals that were still on Mr. Clarke's farm. Mr. Clarke informed me that the first animal which showed signs of disease was the bull Finis Lawrence, which became sick during the latter part of May and was killed in June. The cows Ella Lawrence, Duchess of Broome County, Myrrhine, and Damask all showed signs of sickness about the middle of June. Ella Lawrence was killed with the bull; Duchess of Broome County died. Myrrhine and Damask recovered, and were on the farm at the time the examination was made. Tama Warren had also been killed, and Mr. Clarke insisted that this was because she was worthless as a breeder. Six animals in all had been killed or had died on Mr. Clarke's place since May. According to accounts received from other sources it is probable that Tama Warren and Nutrina of Tunlaw were sick as early as April 6.

At the time my examination was made Damask and Myrrhine were both in very good condition, showing more flesh than one usually sees with Jersey cows. Their hair was smooth and glossy, and, externally, they presented every appearance of perfect health. My examination was made in the pasture field, where, owing to the wind and the disturbance caused by flies, it was not as satisfactory as was desirable. At this time the lungs of Damask showed no signs of disease. Myrrhine showed dullness and loss of respiration behind the right shoulder. Two other cows in the herd and two bulls presented more or less evidences of lung disease, but this was not sufficiently marked to allow a positive conclusion as to its cause. One cow was evidently affected with tuberculosis. Mr. Clarke had brought on his place since June 1 one animal from New Jersey, three which he had purchased at the Epler sale at Viginia, Ill., one from C. A. Keefer, of Sterling, Ill., and several from Wisconsin. It was impossible to judge from any information which I could obtain from him in what manner the disease had been brought to his place. As Ella Lawrence had come from Peoria, and as I heard rumors of disease at that place, I decided to make my next investigation there.

August 16, I called on Messrs. D. H. and S. S. Tripp, and on Mr. O. J. Bailey, at their offices in Peoria. These gentlemen at once admitted that they had lost animals from some disease, the nature of which they did not understand, and they freely placed at my disposal all the information which they could obtain bearing upon the matter. It was here that I gained my first insight into the history of the introduction of the disease into Illinois, and I am greatly indebted to Mr. Bailey and to Messrs. Tripp for the valuable assistance which they gave me. The first cases of this disease occurred in the Tripp herd, and they assured me that the only animals that had been brought upon their place for several months before this sickness were three cows purchased at the Virginia sale, which occurred February 21. These cows were Helena Rex, Albert's Pansy, and Fancy Le Brocq. These animals when first brought from Virginia were taken to Mr. Tripp's stable in Peoria, and afterwards Helena Rex was taken to his farm, which is situated about two miles from the city. I have been told by people who were at the Virginia sale that Helena Rex was coughing at that time and did not appear to be in good health, but Mr. Tripp either did not notice this or was not impressed with the idea that she was affected at all seriously. The first cow that presented unmistakable evidences of disease was Pomare, a cow kept for family use in the town stable. The earliest symptoms were noticed with her about the first of April, and she died April 17. She was treated by the veterinary surgeon for lung fever, but she had previously been a good, healthy cow. Helena Rex aborted April 25, and within three or four days became sick and was treated for inflammation of the womb. She had a cough, but after a few weeks apparently recovered. When I examined her, August 17, there was dullness over a portion of the right lung and a creaking sound heard on auscultation. This lung had evidently been extensively affected, and a considerable part of its tissue had been destroyed by disease. No other cases of disease occurred until July 12, when the cow Anna's Orphan presented the symptoms of inflammation of the lungs, and died July 27. The next case occurred July 25. It was a cow called Queenette, which died August 4. A *post-mortem* examination showed extensive inflammation of the lungs and pleura. The last case which had occurred at that time was a calf, daughter of Pomare, which died August 13 after a short sickness. An examination after death left no doubt that the disease was inflammation of the lungs and pleura. All of these animals had been taken down suddenly with an acute disease, which rapidly ran its course and terminated fatally. I was assured that there had been no disease of this kind among their cattle, nor among any other cattle in the vicinity, so far as they knew, previous to the purchase of the three animals at Mr. Epler's sale.

The first sickness in Mr. Bailey's herd occurred with a cow called Lady Florentia, which had been in his stable in Peoria up to May 10, and was then taken to his farm 7 miles in the country. This cow had not been in actual contact with any of Mr. Tripp's cattle, and the only way in which the disease could be accounted for in her was that it had been carried by some person going from one stable to the other. She showed no signs of disease until about the middle of June. Her trouble was thought to be indigestion. She milked less than usual, but still gave so much that she could not be dried up. She was sick about three weeks, but was in pasture and had a fair appetite all the time. When I examined her, August 16, she still had a bad cough. There was dullness over the anterior and inferior portion of the right lung, with partial loss of murmur and a whistling sound. At that time she was said to be improving in appearance, her eyes were bright, her coat smooth and glossy, and her external appearance was that of good health. The second animal to become sick was Lechene. She showed the first symptoms about the 20th of June. There was loss of appetite and milk, fever, difficult breathing, and cough. She died August 8, and examination showed the right lung solid and red, greatly enlarged, and was described as looking like liver; it was adherent to the ribs and covered with false membranes. The third cow to sicken was Champion De Pansy. The first symptoms were seen August 11 or 12. August 16, when I examined her, there was dullness over both lungs, loss of respiration on the right side, labored breathing, and a temperature of 105⅝° F. August 18 this cow was killed for examination, in the presence of Dr. J. H. Rauch, secretary of the State Board of Health, Dr. N. H. Paaren, State Veterinarian, both of whom I had invited to be present in order that they might see the disease and be convinced of its nature. An examination revealed the right lung adherent to the costal pleura throughout its whole extent, and also firmly attached to the diaphragm; the false membranes were yellowish-white in color, and from one-fourth to one inch in thickness. Two gallons of yellow effusion surrounded the lung. The lung tissue was nearly all hepatized, marbled in appearance, with interlobular connective tissue distended with exudation; the pericardium was thickened and covered with false membranes. The left lung was not hepatized, but it was congested throughout, and the pleura was covered with delicate, false membranes, which were solidly adherent to the costal pleura.

Disinfection was commenced at once at both of these places, and every measure has been taken to prevent the extension of the disease. The cow Lady Florentia was a very valuable animal, but I informed Mr. Bailey it was necessary for the safety of his herd that she should be slaughtered. This has since been done, and I learn that her owner was fully satisfied, from an examination of her lungs, of the importance of this measure. The information which I obtained here made it seem very probable that Mr. Clarke had introduced the disease on his place also with animals purchased at the Epler sale. He received from there the cows Midnight and Nutrina of Tunlaw, and with these was shipped to his place Ella Lawrence, a cow that was sent to the sale at Peoria by Mr. Tripp. According to the best information I could obtain, Nutrina of Tunlaw was the first cow to sicken on Mr. Clarke's farm, and she doubtless was the means of infecting his other animals. The Virginia sale was quite a large one, and animals from it had been sent to a large number of places in different Western States.

The condition of affairs now seemed so very serious that I returned to Chicago, and, with your approval, issued the following circular:

DEPARTMENT OF AGRICULTURE,
Washington, August 20, 1884.

To the Cattle-Owners of the United States:

Owing to the existence of a disease supposed to be contagious pleuro-pneumonia in several herds of Jersey cattle in the State of Illinois, I hereby request owners of all herds of Jersey cattle in the United States, into which new animals have been introduced since January 1, to stop shipments of cattle until after October 1. The disease seems to have been introduced by animals sold at Virginia, Cass County, Illinois, in February, 1884, and these animals were widely distributed through the Western States. It is hoped, therefore, that persons owning cattle tracing to this sale, and all others having cattle affected with disease of the lungs, will at once communicate with Dr. D. E. Salmon, Chief of the Bureau of Animal Industry, care of the Breeders' Gazette, Chicago, Ill., and clearly state the condition of their herds and symptoms of the disease.

The attention of owners of cattle and railroad and other transportation companies is called to section 7 of the act establishing the Bureau of Animal Industry, which makes it a misdemeanor, punishable by a fine of not less than $100 or more than $5,000, or by imprisonment of not more than one year, or both, for shipping cattle affected with any contagious, infectious, or communicable disease, and especially the disease known as pleuro-pneumonia, from one State or Territory into another. The cordial co-operation of State authorities and of all persons interested in the welfare of our cattle industries is earnestly desired in order to avert this danger which now menaces the herds of the country.

GEORGE B. LORING,
Commissioner of Agriculture.

August 22, I visited Mr. Epler's place at Virginia, secured a complete list of the animals which he had sold on the 21st of February, and made an examination of the remainder of his herd. I found no animals there showing any symptoms of pleuro-pneumonia, and Mr. Epler informed me that he had lost none from his original herd since the sale; but a cow called Jessie of Cloverside, which he had bought at Beardstown, Ill., and brought to his place in April or May, died in June of an acute lung disease, which, judging from the description of the appearance of the lungs that I received from him, must have been pleuro-pneumonia. A cow called Deerfoot, which was purchased by Mr. Bevis, of Virginia, at the sale, contracted pleuro-pneumonia and died in March. The description of the symptoms and appearance of the lungs was very clear and left no doubt of the nature of the affection. This animal had not come in contact with other cattle during her sickness. Another cow, called Jennie of Mapledale, was sold to Porte Yates, of Springfield, Ill. She was afterwards attacked with pleuro-pneumonia and died in April. I saw the owner of the cow and the gentleman who made the *post-mortem* examination, and their statements show that she had been affected with pleuro-pneumonia. Maud Holly was sold to E. S. Hodson, of Springfield, and soon after her arrival was treated for some slight sickness. When I examined her, August 22, she was looking well, but had a cough, with slight dullness and loss of respiration over the posterior part of the left lung. Mollie of Mapledale was sold to Frank Gaston, of Normal, Ill. She became sick April 6, a few days after having dropped a premature calf. She was supposed to be affected with lock-jaw, and no *post-mortem* examination was made. It is difficult to say at this time what really was the matter with this cow, but I am inclined to think from the history and symptoms of the case that it was one of pleuro-pneumonia, although no other animals in the herd contracted the disease. The cow Patalene was purchased by D. W. Rawlings, of Jacksonville, Ill., but he left her with Mr. Epler until April 2 or 3. She was afterwards sick, but there is considerable discrepancy in the various accounts I have received as to the character of her trouble and the time when she showed the first symptoms. Mr. Rawlings gave Dr. Trumbower a statement dated September 22, in writing, that he foundered her a little the first time he fed her, and that he called Mr. Rockwell to see her the following Sunday, and that this gentleman gave her medicine on Sunday and Monday and possibly on Tuesday; that she never was sick a day after that to his knowledge. Mr. Rawlings had previously written to me, under date of August 22, that he understood from inquiries recently made that his cow had been affected with lung fever. He knew she had a very bad cough and did not get over it for several weeks. He stated that she still coughed once in a while for a few minutes at a time, as if she were choked. Charles Rockwell stated to Dr. Trumbower that his visit to treat the cow Patalene was on the 10th of May. He knew this from an entry in his book, but he fails to remember anything about her having been overfed. He stated that she had a very bad cough, and he diagnosed the disease as influenza. She recovered in about four days. Soon after that she was turned into Mr. Morrison's field to pasture. On the 10th of June a roan Short-horn cow belonging to Mr. Rawlings was taken sick in the field, and Mr. Rockwell treated her. He could not tell positively what ailed her, but she did not cough as bad as the Jersey cow. He treated her for a month in the stable of Rawlings, and accepted her in pay for the treatment. Mr. Rawlings said that she coughed very badly, and it seemed to hurt her very much. He thought she was taken sick ten days or two weeks after the illness of the Jersey. Mr. Rockwell turned this cow into the Morrison pasture for a while, and then took her to his own barn and kept her there until August 7, and she was killed for beef a few days later. Mr. Morrison had two cows in this pasture. One of them began to cough, and Thomas Scott was called to treat her on the 19th of June. She was sick and weak, though eating all the time, and thin looking; she protruded her tongue while coughing. Dr. Tiffany treated two cows in the adjoining field, which, he states, were affected with simple catarrhal fever. They recovered in a few days. Dr. Trumbower examined Patalene and a cow belonging to Mr. Morrison on the 18th of September, but failed to find any evidence of disease at that time. The cows Julia May and Jane Stoughton were sold to J. B. Warlow, of Danvers, Ill. He took them home a few days after the sale. Julia May was thin and looked bad when he bought her. She had a cough when she reached his place. In the month of June her cough became more frequent, and she lost flesh and strength; her breathing was very difficult, and she died on the 10th of July. They made a *post-mortem* examination and found one lung attached to the ribs and the covering coated with a "scum." This lung was double the weight of the other. In cutting across the solidified portion it had a marbled appearance. Jane Stoughton has never been sick nor even coughed. The cow Eva B. was sold to William Hanna, of Golden, Ill., who received her on the 23d of February. She coughed occasionally at that time, but no attention was paid to it. About one month after the purchase she became sick, refused to eat, and appeared dull and stupid. This continued for several days; she then aborted, and eight or ten days later appeared to be well again, with the exception of an occasional cough. She was examined by Dr. Trumbower in the latter part of September, and at that time presented no evidences of disease.

W. F. Whitson & Son purchased at the Epler sale the cow Pansy Lassie and the bull Andrew. I learned from Mr. Epler and others that Pansy Lassie had an attack of what was supposed to be pneumonia soon after she was received at Virginia. She was treated for this disease, and at the time of the sale was much improved, though the statement was made that she had recently been sick, and she was not sold as a perfectly healthy cow. Mr. Whitson did not understand that she had been sick with lung fever, and though she was thin in flesh he did not suspect any previous disease. Examined September 17 by Dr. Trumbower, she presented the most positive evidences of chronic pleuro-pneumonia. There was very extensive and probably complete hepatization of the left lung; only a very small amount of air entered the larger bronchi in the upper part of the lung and produced a whistling, blowing sound at each expiration. The right side showed no marked symptoms of disease. This cow coughed every ten minutes or oftener when she was made to move. The cough was suppressed and shallow, and apparently accompanied by more or less pain. The bull Andrew appeared to be all right, but two or three other animals in the herd were beginning to cough. William Bell, of Miami, Mo., purchased eighteen head of cattle at the Epler sale. One of these, Zadie, was taken sick about the 12th of May and for ten days ate very little and had a cough. She was isolated, and after calving began to improve and was turned in with the other cattle. This herd was examined August 27 by Dr. Trumbower, when all the animals were found in fine condition, with no signs of pleuro-pneumonia in any. This completes the list of animals sold at the Epler sale, which our investigations show to have afterwards been affected with a disease having symptoms resembling those of contagious pleuro-pneumonia.

As very many of the cattle sold at the Epler sale soon afterwards became affected with pleuro-pneumonia, and as the mingling of the animals at this sale was the only means by which many of these herds could be connected, it became very certain that the disease in Illinois had been brought to that State with some animals that had been sent to Mr. Epler. The animals which he had collected for this sale had come from a number of different herds located at widely separated points. An investigation of the condition of these herds led us to believe that only one had been affected with pleuro-pneumonia. This herd belonged to Mr. C. R. C. Dye, of Troy, Ohio. On the 28th of December, 1883, Mr. Epler purchased five cows of Mr. Dye. The animals were shipped soon after, and arrived at Virginia January 4, 1884. These cows were Mollie of Mapledale and Jennie of Mapledale, both of which came from the herd of James Lyman, of Downer's Grove, Ill., in May, 1883; Fancy Le Brocq, which was bought at Kellogg's sale in November, 1883, and had come from the herd of D. A. Givens, of Cynthiana, Ky.; Albert's Pansy, from the herd of George V. Green, of Hopkinsville, in November, 1882—this cow had not been on Mr. Dye's farm since her purchase; Pansy Lassie had been purchased of John E. Hamilton, in November, 1882.

Mr. Dye informed me that he was in Europe at the time the disease broke out among his cattle. The first animal attacked was Rayon d'Or, a bull, which had not been away from his place since October, 1883. This animal showed the first symptoms some time in February, and died in March. A number of cattle in his herd were affected and some were killed, but he undoubtedly had two different diseases in his herd at the same time—a severe sore throat and pleuro-pneumonia—and it is now a difficult matter to determine which of the animals were affected with the sore throat and which had this in connection with the lung disease. Three calves were killed which Mr. Dye thought were only affected with the sore throat trouble, but Dr. Butler, who attended his cattle at that time, thought they also had lung disease. When I was at the farm I saw one of the animals affected with the disease of the throat, which was suffering very severely, but which had no signs of disease in the lungs. I feel confident, therefore, that Mr. Dye is correct in stating that some of his cattle were affected with disease of the throat only, but this makes it difficult at the present time to decide just how many cases of pleuro-pneumonia occurred in his herd. He lost, altogether, four animals which died and three which were killed previous to my visit. As soon as this trouble was discovered among his animals all sales were stopped, the sick animals were put by themselves on a different farm; the stables were whitewashed and disinfected and every precaution taken to prevent the spread of the disease to other herds, and so far as we have been able to learn no animals in the vicinity of his farm have contracted the disease. An examination of his herd revealed the fact that a number of animals still showed positive evidence of lung disease. An effort was made immediately to raise sufficient money to purchase and slaughter the affected animals, but the discovery of another affected herd in the State destroyed all hope of obtaining sufficient funds for the purchase of all the diseased animals and thus freeing the State from the disease. Mr. Dye, however, was anxious to get rid of the disease in his herd, and consented to have those animals slaughtered which presented the plainest signs of the affection on condition that the State board of agriculture would recommend the granting of compensation at the next sitting of the legislature.

September 19, I selected seven animals which presented signs of lung disease, and these were appraised and slaughtered. The following is a brief statement of the *post-mortem* appearances:

No. 107.—Nearly all of the surface of the right lung attached to the ribs and diaphragm. The lung was one large cyst, in which the hepatized tissue was nearly all disintegrated and broken down into pus.

Bull.—Right lung adherent to ribs and diaphragm. A cyst 3 by 4 inches in diameter contained hepatized lung tissue badly broken down.

Sylvie.—Posterior portion of the left lung atrophied with fibrous degeneration and adherent to ribs and diaphragm.

No. 145.—Left lung adherent to ribs and diaphragm, encysted mass four inches in diameter containing disintegrated lung tissue.

No. 114.—Left lung slightly adherent to diaphragm and ribs, weighing 12 to 14 pounds, containing encysted mass of eight inches or more in diameter and weighing 8 to 10 pounds.

No. 142.—Both lungs adherent to diaphragm and ribs, atrophy and fibrous degeneration of one-third of left lung, two small and encysted masses of dead lung tissue.

No. 41.—Right lung largely adherent, atrophied with fibrous degeneration; in the pleural space between the lungs and firmly attached to the diaphragm was a large cyst full of purulent liquid and having the capacity of about one quart.

The remainder of the suspected animals were placed by themselves at a considerable distance from the others of the herd, and Mr. Dye consented to consider the whole place in quarantine until such time as I could declare all danger to be past.

Mr. Dye does not know how the disease was introduced into his herd. He had purchased a number of grade Jersey cattle in the vicinity of Baltimore and a large number of registered Jerseys from the herd of the late John W. Garrett, of the same county, in November, 1883. The same month he purchased fifteen head of A. M. Herkness, of Philadelphia, a part at private sale, and a part at his auction. Two weeks earlier than this he had purchased a car-load of registered Jerseys at Kellogg's combination sale in New York. All of these cattle were shipped to his farm at Troy. I believe that the disease was brought with the grade Jerseys which had been gathered up in the vicinity of the city of Baltimore. I have been unable to find pleuro-pneumonia in any herds, so far as examined, from which the other cattle came, and it seems very evident that if the disease had existed in Mr. Garrett's herd or among any cattle at Herkness' auction, or at Kellogg's sale, it would have been carried to other places, and we should have found it in many other herds besides that of Mr. Dye and those which had received cattle from him. It seemed probable that among the grade Jerseys there was one which had recovered from the acute stage of pleuro-pneumonia, and which, while appearing healthy, was still able to communicate the disease, as we know is the case with so many of the animals which make an apparent recovery from pleuro-pneumonia. Mr. Dye has only sold a very few animals since the time when the infection was probably introduced among his cattle. With a single exception, I have been unable to find that animals from his herd had carried the disease to other places besides the herd of Mr. Epler. This exception was a lot of three Jerseys sold by Dye to C. N. Mitchell, of Dayton, Ohio, and delivered about February 18, 1884. The introduction of these animals into Mr. Mitchell's herd has been followed by an outbreak of contagious pleuro-pneumonia, and he has lost seven animals from the disease. An inspection of his herd on September 4 showed that five additional animals had been affected, and still showed very evident symptoms. September 20, I examined two other cows belonging to Mr. Mitchell, one of which, an unregistered animal, had a slight crepitation and blowing sound in the right lung. It was my judgment that she had suffered from a mild attack of pleuro-pneumonia, and he informed me that she had been sent to his farm a month or more previous to be bred to his bull. She had been there but a few days when she was brought back to another farm several miles distant. The following is a list of the animals which died in his herd, with date of death: Doe's Blucher, April 27; Donna Daisy, May 1; Vivianetta, May 6; Rapier's Melville, May 28; one, name not known, died May 31; one, name not known, died June 2.

At the first examination of Mr. Clarke's herd we were informed that he had shipped eleven head of cattle in June to H. D. Frisbie, of Cynthiana, Ky., and that on August 9 nine other animals were shipped to the same party. When it became certain that Mr. Clarke's herd had been suffering from pleuro-pneumonia, I telegraphed to the honorable J. Proctor Knott, governor of Kentucky, under date of August 1, that H. D. Frisbie, of Cynthiana, Ky., had recently purchased twenty head of cattle from Clarke's herd at Geneva, Ill., stating that this herd was undoubtedly affected at that time with contagious pleuro-pneumonia, and that I had ordered a veterinarian to examine Frisbie's herd. In view of the enormous live-stock interests of the State, I recommended the prohibition of all movement of cattle from Frisbie's herd until after an examination of their condition was made. Owing to the fact that the first veterinarian asked to make this examination was unable to leave his practice, the examination of this herd was not made until August 29 and 30. At that time Dr. Trumbower made a careful examination of all animals which had been purchased from Clarke, and a number of others in the herd, and reported to me their condition under date of August 30. He was told by Messrs. Frisbie & Lake, who owned the herd of cattle, that they had purchased only fifteen head from Mr. Clarke, instead of twenty, as we had been previously informed. As there has since been a question in regard to the conclusions reached by Dr. Trumbower at the time of his examination, and especially in regard to information which he communicated to Messrs. Frisbie & Lake, I make the following summary of his report, which was written at Cynthiana the day the examination was made, and before any controversy had arisen, and consequently at a time when there could have been no reason whatever for stating anything but the exact truth. There were six calves, as follows: Nora Lawrence, temperature on August 30, 102.6° F., respiratory murmur entirely absent in left lung and complete consolidation revealed on percussion. To account for this condition it was stated that this calf stuck fast in the mud of a pond and nearly suffocated. It was said to have always eaten, and to have shown no signs of sickness except a severe cough. It was more emaciated than the other calves; born some time in March. Floss Lawrence, temperature 104.6° F.; some consolidation in upper portion of the left lung; cough dry and frequent, with mucus and crepitant rales and dry, rasping sounds; born May 4. Flora Bronzo, temperature 103° F.; dry cough, white mucus discharge from the nostril. Fairy Bronzo, temperature 103.6° F.; no symptoms except cough. Cicero Sunbeam, strong mucus rhonchus. Fancy Cruiser, dry wheezing sounds, especially in left side; temperature 103.6° F. All cf these calves had been kept isolated in a pen and fed out of the same pail with a half gallon of skimmed milk twice daily to each calf. They were thin in flesh and all have a cough. They were placed in this pen on their arrival and have not been in contact with the older cattle. The following are the cows which showed signs of disease at that time: Flora St. Hilaire, temperature 103° F.; received from Clarke August 8; dullness over the upper part of right lung. Bell St. Hilaire, temperature 103.1° F.; received from Clarke August 8; did not detect any positive evidence of lung disease, but her breathing was more rapid than other animals in the same condition, and she had a suspicious cough. Flora Orange Peel, bought in Wisconsin in 1883; temperature, 102.5° F. Several small spots revealed dullness over the lungs and others an undue resonance on percussion. Sarah 1st, bought at a combination in New York in May; temperature, 103° F.; cough frequent, dry, and husky. Dora Mellwood, from the same sale; temperature, 103.4° F.; dullness over the lower half of eighth and ninth ribs of left side. Rissa Cicero, bred by Mr. Lake; temperature, 103.8° F.; had a harassing cough. Jessie St. Hilaire, purchased from Clarke August 8; temperature, 103.6° F.; cough dry and frequent; dullness over the lower half of the seventh, eighth, and ninth ribs on right side; pain evinced on pressure on intercostal spaces. Lorne D., received from Clarke July 1; temperature, 104.5° F.; troubled with cough. Many of the other cattle have a dry, husky cough. Frisbie & Lake state that the rag-weed is the cause of this, but it is my belief that many of the animals may be in the initial stage of pleuro-pneumonia. The seventy-one head of this number which were examined were pastured on 160 acres of blue-grass land, having plenty of water and shade. They were in good condition; their coats looked smooth and healthy, with a few exceptions. Frisbie & Lake stated that they had not had a sick animal on their place this season; that they had not sold any animals except twenty-two head which are to be delivered to Dr. Hamilton in the month of November. Dr Trumbower added:

> I told Frisbie & Lake the condition in which I found all their animals, and strongly warned them against disposing of any of them. I told them that Flora St Hilaire, Nora, and Floss Lawrence were evidently afflicted with pleuro-pneumonia, and that several others were exceedingly suspicious. They, however, will maintain the assertion that none of them are diseased, and will abide their time to discover the true nature of the affection. I have good reason to infer from the conversation I had with Frisbie that if any of their stock become actually sick, they will quietly kill them or some accident will happen. They told me they would use every means in their power to remove all suspicion from their herd, and that if any injury was done to them as a result of the investigation they would hold the Government responsible. Mr. Lake thought he would refuse to allow you [Dr. Salmon] to make a second examination, as they were satisfied with the one I made, and that they, if they considered it necessary, would employ a veterinarian, or a number of them, to make examinations on their part, and that they would have to take a stand on the defensive. Mr. Frisbie wanted to make a compromise with me in this way: That if I would pronounce the older cattle entirely free from disease, they would be willing to kill all of the six head of calves, but would not allow any *post-mortem* examination made of them; they would be willing to state that I had pronounced one of them suspicious, and therefore they, to insure safety, had killed the calves and removed thereby all danger of further contamination. I refused to enter into any such compact, but told them that I would report to you the exact condition in which I found their cattle, and that I would not express a positive opinion of the nature of the disease, although the actual condition of two of them, in connection with their history, would be sufficient grounds for a positive diagnosis without any great danger of mistake. They were going to send dispatches to the leading stock papers, making the statement as I made it to them, so there should not be any conflict between them and me, and went so far as to write it down. Nevertheless, after all that promise and seeming show of doing justice to us and to themselves, they forwarded dispatches stating that I found no disease in their herd and pronounced them all healthy.

The following correspondence, which explains itself, shows the action taken by this Department upon the receipt of the above report:

UNITED STATES DEPARTMENT OF AGRICULTURE,
Washington, D. C., September 13, 1884.

GENTLEMEN: I am informed by Dr. D. E. Salmon, Chief of the Bureau of Animal Industry, that twenty head of cattle purchased by you of M. G. Clarke, of Geneva, Ill., left that herd at a time when contagious pleuro-pneumonia existed in it. I am also informed that some of these animals now in your herd, when examined by Dr. Trumbower, an Inspector of the Bureau of Animal Industry, showed very marked symptoms of this disease in its chronic form. Animals showing these symptoms are liable to infect others with which they may come in contact for an indefinite time after they have apparently recovered. I desire to inform you, therefore, that your herd is in danger of being infected so long as animals that have once been affected with pleuro-pneumonia are allowed to remain in it; and also that animals that have come in contact or been exposed to cattle that have once had an attack of pleuro-pneumonia endanger other herds which may come in contact with them.

In this connection I would also call your attention to sections 6 and 7 of "An act for the establishment of a Bureau of Animal Industry," &c., approved May 29, 1884, which makes it a misdemeanor to send animals affected with pleuro-pneumonia from one State into another. In view of the great danger now menancing the vast live-stock interests of our country on account of the presence of this dangerous disease in the West, this Department is determined to use all its influence to secure the prosecution and conviction of all parties who are responsible for the further dissemination of pleuro-pneumonia.

Hoping that you will destroy all animals in your herd that have been affected with this plague, and isolate the remainder of the herd that have been exposed to the contagion for three months after such exposure,

I am, very respectfully, &c.,

E. A. CARMAN,
Acting Commissioner.

Messrs. FRISBIE & LAKE,
Cynthiana, Ky.

On the same day the following letter was addressed to the Governor of Kentucky:

UNITED STATES DEPARTMENT OF AGRICULTURE,
Washington, D. C., September 13, 1884.

SIR: I deem it my duty to inform you that I have been informed by Dr. D. E. Salmon, Chief of the Bureau of Animal Industry, that Messrs. Frisbie & Lake, of Cynthiana, Ky., purchased twenty head of cattle of M. G. Clarke, of Geneva, Ill., at a time when contagious pleuro-pneumonia, or lung plague, existed in his (Clarke's) herd. Further, that Dr. Trumbower, an Inspector of this Bureau, recently examined the herd belonging to Messrs. Frisbie & Lake, and found some of the animals showing very marked symptoms of this disease, in the chronic form. Animals showing these symptoms are liable to infect others with which they may come in contact for an indefinite time after they have apparently recovered.

Shortly after the examination of this herd by Dr. Trumbower, a telegram was published to the effect that our Inspector found all the animals in a healthy condition. This was not true, as both Dr. Trumbower and Dr. Salmon affirm.

We have to-day addressed a letter to Messrs. Frisbie & Lake, requesting them to suspend further sales of cattle for the present, and would be glad if you would do the same. Unless stringent measures are used this destructive plague will speedily extend over the entire country.

Very respectfully, &c.,

E. A. CARMAN,
Acting Commissioner.

Hon. J. PROCTOR KNOTT,
Frankfort, Ky.

The Department was informed by the secretary to Governor Knott that the contents of the foregoing letter were promptly communicated to Messrs. Frisbie & Lake.

The following letter, in reply to the one forwarded by the Department on September 13, was received from Messrs. Frisbie & Lake:

CYNTHIANA, KY., *September 15, 1884.*

SIR: We are in receipt of your communication without date, but, from postmark, mailed at Washington on the 13th instant, informing us that you have been informed by "Dr. D. E. Salmon, Chief of the Bureau of Animal Industry, that twenty head of cattle purchased by you [us] of M. G. Clarke, of Geneva, Ill., left that herd at a time when contagious pleuro-pneumonia existed in it." From card heretofore published by us, a printed copy of which is herewith inclosed, you will find a correct statement as to our purchase of Mr. Clarke, which will show the inaccuracy of Dr. Salmon's information as to our purchases. Mr. Clarke, from whom we bought, says it is not true that at the time our cattle left his herd contagious pleuro-pneumonia existed in it, or that it ever has. You further say in your letter: "I am also informed that some of these animals now in your herd, when recently examined by Dr. Trumbower, an Inspector of the Bureau of Animal Industry, showed very marked symptoms of this disease in its chronic form." By reference to our inclosed card you will see Dr. Trumbower's statement to us of the condition of our herd after he concluded his examination. That statement was prepared in Dr. Trumbower's presence, read to him, and after corrections made by him was said by him to contain nothing but the facts as he found them.

Dr. Trumbower, when he came to our place, was given every facility and our hearty co-operation. He began his examination on the 30th of August and concluded it on September 1. We demanded of him the result of his investigation, and he gave it to us as set forth in our card. From him nor other official of the Government did we receive any further information or communication of any kind regarding our herd. It may be imagined how painful was our surprise to read in the Commercial Gazette of Cincinnati of the 12th instant the following dispatch from its special correspondent:

"LEXINGTON, KY., *September* 11.

"A committee of prominent cattle-men went to Frankfort to-day to endeavor to procure quarantine orders against the herd of Jerseys at Cynthiana, Ky., the property of Frisbie & Lake. The United States veterinary surgeon examined the cattle and found a number of them infected with pleuro-pneumonia in its worst form. Armed with a report to this effect they went to Frankfort, but failed to make anything by it, no Kentucky law applying to the case. The cattle-men will probably have to take the law into their own hands unless the State provides for them. Fears that Ohio and other States will quarantine against Kentucky are expressed, which would seriously affect the market and cut off sale of a large amount of fat, salable cattle here."

Also the following special to the Louisville Courier-Journal:

"FRANKFORT, KY., *September* 11.

"Reliable and satisfactory information having been received by the cattle-breeders of this section of the State that veritable pleuro-pneumonia was now affecting the herd of Frisbie & Lake in the county of Harrison, a large delegation of breeders came to the city this evening to hold a consultation with the governor on the best means of preventing a spread of that dread disease. To give their cause of complaint its due importance, they showed the governor the following statistics: 'From 1711 to 1769 it destroyed 200,000,000 head of cattle in Europe; from 1793 to 1796, from 300,000 to 400,000 in Italy; and in 1842, 300,000 head in Egypt. In 1865 it had proved fatal to 500,000 head in Great Britain in eighteen months.' The same article says, 'the malady is propagated by contagion, that treatment is inefficacious, and that extinction of the poison by slaughter has been shown by the experience of a century and a half to be the only satisfactory and economical method of contending with it.' The attorney-general was present at the meeting, and said that the laws of Kentucky only provided for the impounding of diseased cattle, and could not cover the threatened danger. In the matter under advisement the breeders, among whom were Mr. A. J. Alexander, Mr. Lucas Broadhead, and others of this county—also of Scott, Fayette, and Bourbon Counties—then agreed to arrange for a meeting representing not only the large breeders, but all the cattle owners of the State, to devise ways and means of stamping out the disease and protecting their immense interests from its dread ravages. They ask the press of the State to please take notice of this intention on their part, affecting as it does the price and wholesomeness of beef, and consequently the interest of every individual in the State. A day and place of meeting will be designated later."

And also the following editorial:

"And so it seems, after all denials, that the fearful cattle scourge, pleuro-pneumonia, has broken out in at least one point in Kentucky—in a herd of Jerseys in the neighborhood of Cynthiana, Harrison County. Speedy measures should be adopted to stamp out the scourge, for otherwise it is impossible to form any idea of the ruin that awaits one of the largest and most important interests of the State. The peril of the situation is increased by the fact that Kentucky is without a law efficacious in the case. Already the leading breeders are moving in the matter, and no doubt something to the purpose will be accomplished."

And to find that articles of like tenor were in the Lexington papers. Thus, through the whole State, was disseminated the information upon authority of Dr. Trumbower that our herd was affected with the dread disease, pleuro-pneumonia, creating fear and consternation everywhere, and jeopardizing the values of all the cattle interests of the State. Immediately we telegraphed Dr. D. E. Salmon, inquiring of him what Dr. Trumbower had reported as to our herd, and thus learned for the first time from a Government official that it was reported our herd was infected with pleuro-pneumonia. We cannot conceive what changed Dr. Trumbower's views after leaving here as to our herd having the disease. We do not understand why, after his change of view, we were not notified of it, being the parties most deeply interested; why we were not notified by Dr. Salmon that he had so reported; why some official announcement of the fact was not made to us and the public, instead of seeking an indirect and, to us, unknown channel to reach the public through the press. We feel that we have been unfairly dealt with and may be wrongfully subjected to very great loss.

Thanking you for calling our attention to the law, we would say we have heretofore been law-respecting and abiding, and propose to continue so.

You will readily perceive from our statement as to Dr. Trumbower that we would be compelled to have more reliable evidence than any statement he could make to believe our herd was diseased. We do not believe that any of our herd is affected with pleuro-pneumonia; so believing, of course we will not destroy any of them.

Very respectfully, your obedient servants,

FRISBIE & LAKE.

Hon. E. A. Carman,
Acting Commissioner of Agriculture.

The following is the published card alluded to by Messrs. Frisbie & Lake in the foregoing letter:

NO CATTLE PLAGUE AT CYNTHIANA.

Editor Farmer's Home Journal:

A publication having appeared in the Breeder's Gazette and other papers that a certain lot of Jersey cattle had been shipped from M. G. Clarke, Geneva, Ill., to Cynthiana, Ky., we desire to say that about July 1 we purchased of Mr. Clarke seven registered Jerseys—three cows, one yearling, one last winter's, and two small calves; and about one month afterward received a letter from him stating that he would like to sell us another lot. The first purchase giving the satisfaction they did, we visited Mr. Clarke and made the second purchase of seven head, making in all fourteen head of registered Jerseys, Mr. Clarke putting in a grade heifer to pay freight from Geneva to Chicago, Ill. We received the first purchase with Nutrina of Tunlaw (9946), about the 1st of July, and the second about the 10th of August. Not having any intimation in the least that this stock had been exposed to any contagious disease, and they all being in fine condition, we, of course, turned them in with the balance of our herd, except the four calves, which were turned in a lot with a few other calves. We can state positively that not a single one of our entire herd of nearly eighty head has ever been sick since the Clarke stock was turned in with them, and we can now say, without fear of contradiction, that no herd of Jerseys in the world are healthier or freer from disease than ours. We had one calf that fell into the pond the day after arrival, and, we think, took cold, and has not done as well as the balance, but with this exception have had no sickness whatever, and can say to the world that our herd is entirely free from any contagious disease whatever, and stand prepared with our herd to substantiate this assertion.

Now for the investigation made by the United States Government on August 30 and September 1. Dr. M. R. Trumbower, United States Veterinary Inspector, was ordered to our place to inspect our herd by Dr. Salmon, Chief of the Bureau of Animal Industry, and after making a thorough and careful examination of our entire herd, consisting of seventy-one head of thoroughbred cows and heifers, he found them all healthy and in good condition with the exception of one cow and one heifer that never saw the stock sold by Mr. Clarke to Mr. Keefer and Mr. Boyd, of Illinois. These two, he claims, have affection of the lungs, but would not pretend to state or say that they were affected with pleuro-pneumonia or any other contagious disease. These two, as well as the balance of the entire herd, are now in a fine, healthy condition. Then he carefully examined the calves running in the lot mentioned together. One of these calves he found with left lung consolidated, this being the calf that fell in the pond heretofore mentioned, and one other calf discharging at the nose and a cough, showing evidence of bronchial and lung affection in a slight form. The cough he attributed to eating rag-weed.

After examining and re-examining, this was the extent of his discovery of any disease in our entire herd. Nutrina of Tunlaw (9946), the cow that was said to have taken the disease to Mr. Clarke's place, and was supposed to have been badly diseased with the dreadful disease of pleuro-pneumonia of the worst form while at Mr. Clarke's, proved, upon three close and careful examinations by Dr. Trumbower, to be entirely free from any disease and sound as a bell, with the remark "that she was safe in any herd, and that we need have no fear of her." This cow has been running with our herd over two months. Now, if any breeder of Jersey cattle, with a herd of about eighty head, can say that after as thorough an examination as this he only finds two or three head out of condition he certainly is fortunate. Now, Jersey breeders and the public at large, you need have no fears of any disease from our herd. We propose to stand by them, knowing they will stand by us, for we know their healthy condition substantiates this statement.

In conclusion we desire to correct the statement made in the papers that there were twenty head shipped to Cynthiana, Ky., there being only the number named above. We wish to impress upon the public the fact that not a single head of our herd, including all the Clarke stock, both cows and calves, has ever refused for a single day to take its feed, and certainly this is of itself sufficient evidence of its healthy condition.

FRISBIE & LAKE.

CYNTHIANA, KY., *September* 1, 1884.

Three days later Messrs. Frisbie & Lake addressed the following letter to the Department:

CYNTHIANA, KY., *September* 18, 1884.

SIR: Since writing you on the 15th instant the following facts have occurred, which we deem it proper to advise you of. On Sunday last we observed an old cow of our herd was off and droopy, showing signs of sickness. There was nothing, however, in her appearance to excite alarm, nor were her symptoms those commonly attributable to cattle affected with pleuro-pneumonia. However, on yesterday the cow became very sick and much bloated. We relieved her by puncturing the stomach, allowing the gas to escape. But as she was evidently a very sick cow we immediately telegraphed to Dr. E. T. Haggard, veterinary surgeon at Lexington, to come here this morning. In response to our summons he came. The bloat of the animal prevented his making such examination of the lungs as would determine the extent of their affection, if affected at all, and he asked that the animal might be slaughtered and a *post-mortem* examination made. To this we readily consented, and the animal was killed and the examination made in our presence. The right lung was evidently very much diseased, and Dr. Haggard pronounced the disease with which the animal was affected as pleuro-pneumonia. Another animal in the same pasture is sick and seriously affected as the cow we slaughtered, and will probably die. Two or three cattle on another place are not well. Our herd consists of about two hundred and fifty head, and all of them had an opportunity to take the disease if contagious.

Very respectfully, your obedient servants,

FRISBIE & LAKE.

Hon. E. A. CARMAN,
Acting Commissioner of Agriculture.

The meeting of stock-men, alluded to by Messrs. Frisbie & Lake in their first letter to the Department, was held in Lexington, Ky., September 19, 1884. The Department is indebted to Mr. D. Runyon, secretary, for the following official copy of the proceedings:

PLEURO-PNEUMONIA IN KENTUCKY.

In response to an urgent request of the president of the Kentucky Shorthorn Breeders' Association, a large number of Jersey, Shorthorn, and grade cattle-breeders assembled at Lexington, Ky., September 19, 1884, to determine on the course of action in regard to the report officially made that pleuro-pneumonia existed in the herd of Jerseys owned by Messrs. Frisbie & Lake. The Rev. Rutherford Douglass, a prominent Jersey breeder, was called to the chair, and Mr. Dan Runyon, of the Live Stock Record, was made secretary. Messrs. Frisbie & Lake did not arrive until the meeting had made some progress, but were represented by their attorney, Hon. Caleb West. Mr. Estill stated that the object of the meeting was to determine whether pleuro-pneumonia did or did not exist in this section, and that as it was a subject of vital import to cattle-men, we should at once take action on the affair. The chairman responded that the disease was the most destructive known, and that everything known of its existence in the State should be fully told. The secretary was then handed the following letter to read:

[This was the letter addressed by the Department to Governor Knott on September 13, 1884, quoted above.]

Several short speeches were then made by Attorney-General P. W. Hardin, Dr. Keller, Leslie Combs, Ethelbert Warfield, and others.

Mr. Henry Higgins said he would like to hear from the other side, and Judge West responded that all would be told in good time; that he would give everything that his clients knew, and stated that the first intimation they had of the existence of pleuro-pneumonia in their herd was a special dispatch sent to the Commercial Gazette. They expected to prove that Dr. Trumbower was unworthy of credence, as he made a statement in the presence of responsible parties to the contrary of the statement made therein.

Capt. Phil Kidd wanted to read a dispatch from a Nashville paper which charged pleuro-pneumonia on the cattle sold by Mr. Alexander McClintock at a recent sale. Mr. McClintock made a short but satisfactory speech, and when the proposition was broached offered to pay the expense of a committee going down to examine his herd, and part of the expense in killing any of the suspected cattle. This statement was received with much applause. Mr. McClintock, in response to a question, said that one of his cows was sick at Mr. Henry Higgins', and that gentleman referred to Dr. Haggard, who said he would delay his answer until later.

After further discussion the reports were traced through various sources to Dr. Trumbower, Government Inspector, who, on September 1, finished the inspection of Frisbie & Lake's Jersey herd at Cynthiana. Frisbie & Lake were ready to produce witnesses that Dr. Trumbower's conversation thereon during and immediately after the inspection induced them to believe that their herd was free from the disease, and that their card of denial was written in the presence of, read by, and corrected, in one instance, by Dr. Trumbower. On September 13 they saw a Lexington dispatch in the Cincinnati Commercial Gazette, giving Dr. Trumbower as authority, that four cases of pleuro-pneumonia existed in their herd. They telegraphed Dr. Salmon, Chief of the Bureau, who answered that such was the report. They then telegraphed for a copy of Trumbower's report, but never received a reply till September 15, when they received a communication from Mr. Carman, Acting Commissioner of Agriculture, mentioning a report from Dr. Salmon as explaining the introduction of the disease into their herd by twenty cattle bought from Clarke, of Geneva, Ill. But Salmon should know, and Trumbower had been told, that they only bought fifteen from Clarke, who denied the existence of the disease in his herd, and they could not understand why Trumbower should tell two different tales, or why they had received no official notification. They still refused to believe the disease was in their herds. They promptly answered Salmon's letter, also one from Governor Knott requesting them to make no sales from their herd. On Sunday they were notified that two ailing cows had been found. On Tuesday they had several cattle-men look at them, and were still without assurance of pleuro-pneumonia. Wednesday the cows grew worse, and they telegraphed Dr. Haggard, who arrived Thursday morning, held a *post-mortem* on one of the cows killed for the purpose, and found one lung completely diseased and the other touched by undoubted pleuro-pneumonia. He declared the other cow affected in the same way. They at once wrote the facts to all concerned, and stated them fully to the meeting. Dr. Haggard detailed the result of his examination, and showed pieces of the diseased lung. Another cow from Clarke's herd had the disease, and several others showed symptoms.

The following resolution was offered by W. P. Hardin, attorney-general, and unanimously passed:

"*Resolved*, That the statements of Messrs. Frisbie & Lake, with the accompanying papers and proof, we regard as satisfactory and conclusive of their honest, just, and gentlemanly conduct, both to themselves and the public, in regard to the presence of pleuro-pneumonia in their herds; and, appreciating their manly course, we thank them for the cordial and interesting information, as well as their expressed desire to fully co-operate with us."

The following resolution was offered by Mr. Leslie Combs, and passed unanimously:

"*Resolved*, That whereas a Government Inspector examined the herd of Frisbie & Lake on August 31, and since he reported that pleuro-pneumonia existed in said herd, that this meeting condemns his concealment of the results, reported to the governor by Messrs. Frisbie & Lake, whereby they could not sooner have isolated their herd, and that a copy of said resolutions be forwarded to the Commissioner of Agriculture."

The following resolution, offered by Mr. W. W. Estill, was carried:

"*Resolved*, That it is the sense of this meeting that the governor at once call the legislature together to devise ways and means for exterminating pleuro-pneumonia as demonstrated by *post-mortem* to exist in our State."

The following resolution, offered by Mr. T. C. Anderson, was carried:

"*Resolved*, That the governor of Kentucky be requested to call upon the Commissioner of Agriculture of the United States to put into quarantine all the herds of cattle in this State suspected of being infected with pleuro-pneumonia."

Senator John S. Williams said the governor ought to be requested to call the legislature into extra session to consider the matter, and if such session is not called the cattle should be at once slaughtered, and he felt sure that the next legislature would pay the owners a reasonable price for them. He said that if something is not done to stamp out the disease it will destroy the cattle interests of the State.

The following resolution was offered and adopted:

"*Resolved*, That Messrs. Frisbie & Lake and the Chair appoint a committee to devise ways and means to exterminate the disease."

The Chair appointed the following committee in conformity with this resolution, viz: General P. W. Hardin, A. J. Alexander, Leslie Combs, William Warfield, and D. A. Givens.

The meeting adjourned to meet again in Lexington on Saturday, September 27, 1884.

R. DOUGLASS, *President.*

D. RUNYON, *Secretary.*

September 24 I visited and inspected this herd. I examined particularly the animals Dr. Trumbower reported to be diseased, and was able to confirm his report in every respect. At that time a number of additional animals were suffering from pleuro-pneumonia, and several seemed to be in the first stages of this disease, but owing to the great number in this herd, and the large pastures in which they were running, I was unable to make a careful examination of all the animals.

October 3, Dr. Paaren, State Veterinarian of Illinois, found a Jersey heifer at Saint Charles, Ill., which belonged to W. A. Stewart, and had been purchased from Mr. Clarke, that was suffering from pleuro-pneumonia; also a ten-year-old native cow, belonging to the same gentleman, which had been running with this heifer and had contracted the disease from her. Both were slaughtered, and a *post-mortem* examination is said to have revealed typical pleuro-pneumonia. The following week Dr. Paaren killed a second Jersey heifer at Saint Charles, which belonged to D. B. Moore, which had been purchased early in the spring from the Clarke herd at Geneva.

The following is a recapitulation of the total number of animals affected in this outbreak according to our information:

Owner.	Number exposed.	Number affected.
C. R. C. Dye	65	20
C. N. Mitchell	30	13
W. C. Clarke*	34	17
John Boyd	21	13
D. H. & S. S. Tripp	65	7
O. J. Bailey	60	5
A. G. Eplert†	100	8
D. W. Rawlings	1	1
W. A. Stewart	....	1
Frisbie & Lake	250	16
Total	626	101

*Includes the animals sold which were afterwards found diseased.

†About the number exposed previous to the sale, and the number affected includes those which contracted the disease after the sale.

CHARACTERS OF PLEURO-PNEUMONIA AS REVEALED BY THE HISTORY OF THE DISEASE IN EUROPE.

There seems to be a very general misapprehension, even among the best-informed people of our country, in regard to the characters and results of pleuro-pneumonia in Europe as demonstrated by the history of the disease there; and many of those who are laboring under such misconception of the facts contest the accuracy of the diagnosis of those veterinarians who have asserted that a disease identical with the pleuro-pneumonia of Europe exists in the United States. The grounds upon which the existence of this disease here is contested are principally the following statements: The pleuro-pneumonia of Europe is said to spread with incomparably greater rapidity than our disease; it is more plainly contagious; it attacks a larger proportion of the exposed animals, and is much more virulent and fatal. In the present emergency, when it is so necessary that our people should be correctly informed, and placed beyond the possibility of being deceived by those pretentious persons who, though ignorant of the disease both in Europe and America, still speak with the assurance and certainty of the best informed, I have decided to enter somewhat into detail in regard to the conclusions to be drawn from European history on the points mentioned. Much of the misunderstanding and confusion of ideas in the United States, relative to the characters of pleuro-pneumonia in Europe, results from a failure to discriminate between the different diseases which have decimated the herds of both Great Britain and the Continent. Not knowing the characters by which scientific men distinguish such diseases from each other, and with an imperfect knowledge of the language, Americans have written learnedly of pleuro-pneumonia when they were really dealing with outbreaks of rinderpest or even of foot-and-mouth disease. It would be impossible for an educated veterinarian to make such absurd blunders, but we have not yet passed through the period of our history when people of other professions assume that the veterinary profession is ignorant even of its own special subjects, and attempt to educate it with the valuable information which they from time to time collect during the leisure moments of a few weeks' journey abroad. In time it will doubtless be discovered that our veterinarians do have some knowledge of the diseases of other parts of the world; that their means of distinguishing between them are more reliable than has been admitted by their would-be instructors, and that the knowledge and experience obtained by constant studies of a subject is a safer basis for an opinion than superficial investigations made as a pastime by those who, ignorant of the matter at the beginning, are so foolish as to believe themselves experts after a few hours' training.

HISTORY OF PLEURO-PNEUMONIA IN CONTINENTAL EUROPE.

The references to pleuro-pneumonia in the literature of the past, which are sufficiently definite to make it at all certain that this disease was alluded to, extend no farther than two centuries ago. The passages in the writings of Aristotle, Livy, Virgil, and others supposed by some to refer to this disease, are too indefinite to be seriously accepted as showing its existence before the beginning of the Christian era. We must come down to 1693 before we find an account of an epizootic cattle disease that seems to indicate contagious pleuro-pneumonia. In that year Valentini saw and briefly described a disease which destroyed many cattle in Hesse. He says:

> The preceding winter being wet, but towards the close very cold, at the beginning of spring an unusual heat set in, and continued throughout the whole summer; which sudden change produced an unequal and unnatural motion of the humors and breath, followed by death to man and beast; oxen and cows succumbed in numbers. Amongst other causes, a corrosive dew, which spotted linen with marks more or less dark-colored, and corroded everything, was supposed to produce ill effects. From the observations of the butchers, it was proved that these animals died from pulmonary phthisis (*phthisi pulmonali necabantur*), to which, without doubt, the severe cold after the intense heat much contributed. At the end of July and the beginning of August, besides dysentery and malignant fevers, a certain intermittent fever, like tertian fever, attacked man. (Quoted by Fleming in Animal Plagues, i, 165.)

Delafond said in regard to this observation that—

> Nothing could authorize one to say that Valentini described the pleuro-pneumonia which attacks the cattle of to-day. (O. Delafond, *Traité sur la maladie de poitrine du gros bétail connu sous la nom de péripneumonia contagieuse.* Paris, 1844, p. 19.)

Gamgee, on the other hand, says that while—

> Valentini committed the common error of attributing the lung plague to the weather, his reference to a widespread pulmonary disorder among cattle is sufficient to warrant our dissenting from Delafond, &c. (Report on Diseases of Cattle in the United States. Department of Agriculture, 1871, p. 8.)

In 1713 and 1714 Wirth says contagious pleuro-pneumonia certainly prevailed in Suabia, and also in several cantons of Switzerland. (*Lehrbuch der Seuchen und ansteckenden Krankheiten der Hausthiere.* Zurich, 1846, p. 298.)

Jacob Scheuchzer described a disease which affected horned cattle in the canton of Zurich, Switzerland, in 1732, which he called *gangrène, volante* of the lungs, which was prevalent again in 1743. (Delafond *loc. cit.*, p. 20. Wirth, *loc. cit.*)

In 1765 Bottani says gangrenous inflammation of the lungs affected cattle in the Tyrol. (Fleming, An. Pl., i, 424.)

These and other references more or less incomplete and unsatisfactory of themselves, prove very little in regard to the real nature of the disease which caused the ravages referred to; but in connection with the results of later investigations they appear to show that the mountains of Switzerland and adjoining countries had long been infected with this disease.

In 1769 there was an extensive epizootic in the north of France which was investigated by Bourgelat, the founder of the first veterinary school in Europe. There seems to have been a disease of horses and cattle at the same time which was considered by some to be identical, though the works which I have been able to consult do not make it certain that Bourgelat entertained such an opinion. The cattle disease existed in Franchl-Comté, Champagne, and Hainault, and was said at that time to appear annually in Franche-Comté, where the people called it *murie*. Nearly all French veterinary writers accept the disease called *murie* as being identical with the contagious pleuro-pneumonia of the present day. Fleming takes an entirely different view. He says:

> Lafosse (*Traité de Pathologie Vétérinaire*, iii, 616) positively asserts that this "murie" was the bovine contagious pleuro-pneumonia, but surely he must be mistaken. *Horses*, as well as cattle, were attacked, and the proportion of recoveries by medical treatment in this epizooty is never reached in the deadly lung disease of our days. (An. Pl., I, 430, foot-note.)

The statements upon which this judgment is based are that at Avesnes the disease was reported to have broken out first among the horses of two regiments of dragoons stationed there, and from thence it extended amongst the cattle in the election of Joinville. There is, however, no evidence beyond a mere supposition that the cattle and horses were affected by the same disease. Even at the present day it is very common for people, when an epizootic is raging among any kind of animals, to attribute all other diseases that arise among other species of animals, or even among men, to the effects of the one malady. We saw this particularly during the great epizootic of influenza in 1872, and we frequently hear the same thing in connection with swine plague. As we go back nearer to the time when science had not yet furnished the data for distinguishing between diseases, such confusion becomes more common, and it is not surprising that an enzootic of influenza or typhoid pneumonia among horses occurring at the same time as an outbreak of pleuro-pneumonia of cattle should at that period have been considered substantially the same disease.

The statement in regard to the curability of the disease was that the veterinary school of Alfort having been consulted, dispatched some of its pupils to the districts where the disease was most deadly. The measures they proposed had the happiest results, for whereas before the arrival of these men the animals were dying in crowds, they were now able to save 140 out of 160. There is something incredible about this statement, no matter what disease it is applied to, for there is certainly no epizootic disease with which animals are affected that is so fatal and at the same time so amenable to treatment. The probability seems to be that these young men arrived on the field after the first force of the epizootic had spent itself, and that they found a large number of affected animals in the chronic stage of pleuro-pneumonia which were greatly benefited by the simple stimulating and tonic treatment that was used. Delafond says:

> The symptoms noted by this celebrated man, the morbid lesions which he mentions in the lung and the pleura, the causes that he assigns to the disease, the curative measures that he counsels to combat it, demonstrate positively the existence of an acute inflammation of the pleura and of the lung. (*Loc. cit.*, p. 20.)

He goes on to recapitulate the work of Bourgelat as follows:

> *Symptoms.*—Cough more or less dry, not frequent at the commencement but more repeated towards the last. Very marked fever; great depression, above all after eating; distaste for food; suspension of rumination as the disease progresses; offensive breath; dryness of muzzle; sometimes thick, white discharge from the nostrils.
>
> *Prognosis.*—The depression, weakness, the great difficulty of breathing, the continual cough, the redness of the eyes, the dryness of the tongue, the rattling in the throat, the offensiveness of the breath, are fatal signs, as the absence of these same symptoms is reason to hope for cure.
>
> *Morbid lesions.*—Lividity, engorgement, ecchymosis of the lungs, adherence of these organs to the pleura, gelatinous layers of diverse colors, adhering slightly to these organs, redness, thickening of the pleura, considerable effusion of a reddish, foamy, bloody or purulent water in the thorax.
>
> *Causes.*—Atmospheric variations, cold and abundant rains to which the animals are exposed, sudden passage from a warm stable to such rains. Bourgelat does not mention contagion.
>
> *Treatment.*—Abundant blood-letting the first, second, and third day, when the blood which is drawn does not coagulate it indicates the uselessness of this operation, emollient injections, cooling drinks, emollient electuaries, emollient fumigations of the nostrils. This is the treatment counseled for the first period. When the disease is advanced, Bourgelat prescribes cinchona, rejects bleeding, and completes the cure with purgative injections.
>
> *Preservative means.*—Mild bleeding, dieting, emollient injections, *eau blanche*, fumigation with the vapors of acetic acid in the stables (pp. 20, 21).

Of this account Delafond says:

> This succinct analysis demonstrates that Bourgelat saw pleuro-pneumonia and studied it well; and certainly this account is much preferable in many respects to the work on pleuro-pneumonia which was published twenty-two years later by Chabert. To Bourgelat, then, belongs the first description which was properly made of the pleuro-pneumonia of bovine animals.

In spite of Fleming's incredulity, it would appear that this *murie*, which in 1769 had already become domiciled in Franche-Comté, and which was investigated there at that time by Bourgelat, was the same disease which is now known as the contagious pleuro-pneumonia of bovine animals. This conclusion seems the more reasonable because of the other and preceding accounts of similar disease in the neighboring cantons of Switzerland, and because later observations indicate that the disease in other parts of Europe is traced to cattle from this mountain region.

Franque shows that from 1772 to 1830 bovine epizootic pleuro-pneumonia was scarcely ever absent from the Duchy of Nassau. (*Geschichte der Hausthierseuchen im Herzogth. Nassau.* Frankfort, 1834. Quoted by Fleming in An., Pl., i, 456.)

In 1773, Haller published his investigations of an epizootic cattle disease in Switzerland, which he supposed was cattle plague, but which we now know from his careful descriptions was nothing else than pleuropneumonia. This author made some original and most important observations in regard to the disease. He noticed that an animal might be taken from an infected stable and not become sick until a month after its removal, and that it then perishes from the veritable contagion which without doubt had been concealed for the whole of this month in its body. He observed that diseased cattle move about with vivacity for some weeks, giving their usual quantity of milk and retaining their appetite. The only sign of pneumonia noticed from the commencement is a slight cough which affects the animal notwithstanding all the apparent signs of good health. It is not for some weeks after the beast has become infected that fever shows itself by shivering and erect hair. The cough now augments, the animal moans, its strength diminishes, it cannot stand, and lies very often; it has a difficulty in breathing; the pulse is frequent; the heat and fever become intense. Before death a diarrhœa of a bad odor with rapid sinking terminates the animal's days. This diarrhœa does not always occur. When opened after death the lungs are found constantly and infallibly attacked. In all the contagions which have reigned at Sulens, Grandson, at Crassy and elsewhere, the lungs have always been inflamed, and attached to the pluræ. He found the same observations in the best authorities who had written on the contagion, and particularly in the writings of M. Bourgelat, who has made the curing of these animals a particular study. A long experience had taught him that remedies were useless in this disease, and that their administration was dangerous, for the infection is really communicated by the breath and exhalations, and these may lodge in the clothing of those who go about the animals and be conveyed to others yet in health. In his recommendations for prevention he begins by disabusing the public of the idea that the pneumonia (*la pulmonie*) is not a contagious disease. This outrageous idea even comes from some *savants*. It was certain in that country that as often as the disease manifested itself, it was found, when traced to its source, that a beast had been purchased in the market of some suspected place or had been brought from some infected locality and had carried the contagion with it to the new center. Sometimes the native cattle had been pastured with those of a neighboring infected country. A better proof that the pneumonia is perpetuated by infection is the manner in which we can confine it in suspected places by cutting off all communication between the infected and the healthy stables. This contagion does not spread very far, and it does not infect a column of air for any great distance. He then goes on to show that they could only be protected from the disease by prohibiting the entrance of cattle from countries in which it exists. He recommends the disinfection and closing of stables for at least three months after the last case of disease before fresh animals are admitted, and believed that all the animals exposed, whether sick or well, should be destroyed. (Fleming, An. Pl., i, 446–460.)

It will be seen from this brief recapitulation that Haller's work was far in advance of his time. He recognized the contagiousness of the disease, its long period of incubation, the fact that the contagion was not carried far through the air, but could be traced to animals or men who had been in contact with sick cattle. He does not appear to doubt that the *murie* studied by Bourgelat was the same disease which he had seen; and the measures of prevention which he preferred can scarcely be improved upon at the present day. His work is by far the most able and important one which was published in regard to this disease during the eighteenth century.

In 1778, "according to Kausch, bovine contagious pleuro-pneumonia prevailed as a veritable plague in Upper Silesia," and continued until 1790.

It prevailed at the same time in the kingdom of Würtemberg; the commonalty of Tuttlingen, in 1777, had lost about 150 cattle from the malady (KAUSCH, *Originalbemerkungen über die Rindviehsterben*. Leipsic, 1790. WIRTH, *Lehrbuch der Seuchen und ansteckenden Krankheiten der Hausthiere*. Zürich, 1846. Quoted by Fleming in An. Plag., i, 490.)

This disease also prevailed in Istria during 1779 and 1780, as stated by Fleming on the authority of Orus and Bottani (*loc. cit.*, i, 490.)

Dating from the year 1765 until that of 1792, pleuro-pneumonia appeared to attack only the cattle of the mountainous countries, like Switzerland, Jura, Dauphiné, Vosges, Auvergne, Piedmont, Upper Silesia, &c. It had been seen, it is true, to show itself all at once in a few localities, as in Champaign (1769 and 1776), in Bourbonnais (1788); but it is necessary to come down to the years 1789, 1790, 1791 and 1792 to see pleuro-pneumonia abandon the mountains, spread itself over the plains of the whole of Europe, and determine mortalities of greater or less extent. It is worthy of remark that the French revolution began during this epoch, which, as is known, was soon followed by European wars. Then numerous herds of cattle descended from the mountains to form supplies for the armies; but the cattle plague, the faithful companion of great movements of troops, attacked and decimated these animals not only in the camps but also on the farms and everywhere where the convoys of the enemy brought and scattered it. It is also from this epoch—and this is an important circumstance to relate here—that the great agricultural improvements are dated; the crossing of the different races of bovine animals in the mountains with those of the plains; the numerous changes of cattle brought about by the trade between the sections where they were bred and the better agricultural sections where there was abundant pasture used for fattening, and for the production of milk, butter and cheese. Are not these European wars, then, these general and destructive epizootics, these improvements in agriculture, this traffic in animals between the breeding sections and the better agricultural communities, these speculations carried on in the products of milch cows, the causes which have brought the pleuro-pneumonia, which was originally confined to the mountains, into the plains and the countries of improved agriculture in all parts of Europe? I shall attempt to prove that this supposition is not without some foundation.

It is known that Piedmont and Italy were the theater of a part of the wars of the Republic, the Consulate and the Empire, and that the cattle of these beautiful and rich countries have not only furnished the greater part of the supplies of the different army corps which traversed the whole of Italy, but besides this they were ravaged by the cattle plague. Very well; it was at this same time that pleuro-pneumonia descended from the mountains to attack the animals of the plains, the valleys, the suburbs of the great cities. The observations and writings on the pleuro-pneumonia by Brugnone and Toggia in 1792, Roland in 1806, Luciano, Lessonna, Ferrari, Robecci, and Perrotte, from 1826 to 1832, among the animals of the territory of Turin, prove that this is the case. And, to-day, if I am well informed, pleuro-pneumonia still continues its ravages among the cattle of Piedmont.

In Switzerland, pleuro-pneumonia appears to have existed from time immemorial, in the mountains, at least; it was described there in 1732 by Jacob Scheuchzer under the name of progressive gangrene of the lung, and it raged there still in 1743. In 1812, it was studied there by Ythen; in 1823, by Wirth; in 1824, by M. Favre; and in the course of this year (1844) the Sardinian Government has just prohibited the introduction of all kinds of live-stock coming from Switzerland—prohibition which was based on the existence in that country of bovine pleuro-pneumonia.

As in France, the cattle of Germany, and particularly those of Hesse, Swabia, and Upper Silesia, appear to have been attacked at certain times by pleuro-pneumonia; but according to the descriptions which have been left by Valentini (1693), Bucard-Mauchard (1745), and Kausch (1778 to 1784), this disease seems to have been complicated with putridity. But it was during the wars that Austria and Prussia sustained against the Republic, the Consulate, and the Empire, that pleuro-pneumonia appeared and spread itself in almost every part of the country beyond the Rhine. In fact, this disease was observed by Kolpin, in Prussia, in 1800; by Ammann, in Bavaria, in 1808; by Sander, in Prussia, in 1810; by Busch, in Hesse-Cassel, in 1816; by Lappe, in Hanover, in 1818. And since these unfortunate periods, Dieterichs mentions it, in 1821, in Prussia; Waldinger, in Austria, in 1822; Planck, in Bavaria, in 1824; Noetel, in Prussia, in 1828; Merk, in Bavaria, in 1830; Wagensfeld, in Prussia, in 1832; Gerlach, in the principality of Anhalt-Dessau, in 1835; and, besides, if the general veterinary report of the college of medicine of Rhenish Prussia, for the years 1829, 1830, and 1831 is consulted, it is seen that this disease had not left the stables in the vicinity of Cologne, Coblentz, Trèves, Bonn, Bittburg, Dhaun, Adenau, Zeel, and several other districts, and it was from these provinces, near the frontier of the Netherlands, that in 1832, according to the learned Professor Numann, pleuro-pneumonia was propagated among the bovine animals of Guelders, and from this point to all the Kingdom of Holland, which had been up to that time free from pleuro-pneumonia. (Delafond.) *Traité sur la maladie de poitrine du gros bétail connu sous le nom de péripneumonie contagieuse.* 1884, pp. 22–24.)

In 1790, pleuro-pneumonia was epizootic among the cattle of Bavaria, and it prevailed for a considerable distance around Paris from 1790 to 1794. (Fleming, An. Pl., ii, 531.)

In France, with very few exceptions, pleuro-pneumonia was confined to the mountain districts up to 1792. It had made some progress in Champagne in 1769, and in Bourbonnais in 1788; it had also become seated in the dairy stables surrounding Paris in 1790, but the great agricultural districts of France were free from it at that time. In 1796, this disease was so destructive in the province of Luxembourg that it attracted the attention of the Government and Huzard and Desplas were commissioned to study it and arrest its extension. At the same period Dr. Gastellier was trying to limit its ravages in the vicinity of Montargis. From that time to 1810 the only newly infected locality reported was the district surrounding Valenciennes. (Delafond, *loc. cit.*, 28.)

In 1800, epizootic pleuro-pneumonia was stated by Bottani to be generally prevalent in Italy. Fleming thinks, however, that this may have been cattle-plague.

Before 1822, according to M. Tisserant (*Journal de Méd. Vét. de Lyon*, second series, iii, 402), bovine contagious pleuro-pneumonia was entirely unknown in the department of Ardèche, France; but in this year it made its appearance there. It would seem that at this time oxen were, for the most part, employed to tow the boats trafficking on the Rhone, between Lyons and the sea, and that the majority of these animals came from Franche-Comté. In winter, when the river traffic was less active than at other seasons, these bullocks were depastured on the mountains in the neighborhood. In 1822, a certain number of them so depastured were discovered to be diseased, and these propagated the malady. Nevertheless, it being winter, and the native cattle nearly all kept housed, without much communication between the stables, the contagion did not spread to any great distance, and was not long in being eradicated. It appeared again, however, in 1847. (Fleming, An. Pl., ii, 88.)

This disease appears to have been entirely unknown in the department of the Nord, France, until 1826. According to M. Lecoq, it owed its introduction to the following circumstance: This rich department, in order to have its surplus forage profitably utilized, purchased every year in Franche-Comté numerous droves of cattle, and as the disease was present in that region, these droves carried the deadly contagion from their native mountains. For several years it had only affected cattle which were strangers to the department; but the disease gradually spread among the indigenous herds, and is now enzootic among them. It has occasioned most serious losses. (Quoted from *Jour. de Méd. Vét. de Lyon*, second series, iii, 402, by Fleming in An. Pl., ii, 123.)

From 1820 to 1827 there was a remarkable extension of pleuro-pneumonia in the mountain districts of France. It was mentioned by Sajous in the Pyrenees in 1820; in the mountains of the department of Rhône by Grognier in 1821; in those of Dauphiné by Bragard and Michalon in 1823; in Doubs and Jura in 1827 by Tissot. It penetrated to Meuse in 1831; to Bas-Rhin in 1834; to Pas-de-Calais and Nord in 1835; to Côte-d'Or, Aube, Oise, and Seine-et-Oise in 1837; to Nièvre and Seine-Inférieure in 1839. In 1840 it had just attacked the cattle of Seine-et-Marne, Yonne, and Moselle. While the provinces which bred cattle and into which no foreign animals were brought, like Basse-Normandie, Bretagne, Vendée, Anjou, Limousin, Mans, and many of those in the south of France, remained free from the disease. (Delafond, *loc. cit.*, 28, 29.)

Fleming says that during the ten years subsequent to 1827 the disease (pleuro-pneumonia) had extended to and fixed itself in the districts of Merv, Noailles, Beauvais, Abbeville, Arras, Amiens, and Lille. From the year 1831 the malady was particularly well known in France. (Animal Plagues, ii, 136.)

In Belgium M. Gluge stated that it had existed since 1828. He says:

> But in a communication from the late inspector of Belgian cavalry, Verheyen, we find that this insidious plague was imported into Belgium so early as 1827, and proved a dreadful infliction on the Belgian farmers, traveling from place to place, until the whole of Flanders was invaded by it (Fuchs). It has been remarked that no country ever suffered so much from this malady as Holland. It received the infection in 1827—before the separation between it and Belgium—in the, at that period, southern provinces of the kingdom, particularly in Brussels, Mechlin, Louvain, and Diest. At a later date showed itself in Hainault and West Flanders; and in the year 1833 it appeared in all its malignity in what we now call Holland. In that year it broke out on a farm in Guelderland, and from this center it spread over the whole country. (Fleming, *loc. cit.*, ii, 141.)

Dr. Willems, of Hasselt, Belgium, writes:

> This disease was introduced to us from Flanders in 1836, by some beasts purchased of the merchant Moras, and first brought to my father's stables, and to those of M. Platel, distiller. From that time to the present (1853), all the distillers have suffered considerably by it, and many small farmers have been entirely ruined. The disease, at first epizootic among us, has become enzootic, and decimates a considerable number of beasts of the bovine race every year. (Quoted by Fleming, An. Pl., ii. 291.)

RATE OF EXTENSION OF PLEURO-PNEUMONIA IN EUROPE, AS SHOWN BY THE ABOVE HISTORY.

It will be seen by the facts which have been related above, as they are given in the most authentic histories of bovine contagious pleuro-pneumonia in Europe, that the disease there has scarcely spread at all except by the movement of cattle. It existed in Switzerland and Suabia as early as 1713 and 1714, and in 1769 it had been in Franche-Comté so long that it was spoken of as occurring regularly; yet, the only instance which occurred of its spread in France for twenty years was the outbreak in the adjoining district of Champagne in 1769 to 1776; it was carried to Bourbonnais in 1788, and reached the cow-sheds in the vicinity of Paris some time before 1790. Practically, pluero-pneumonia is known to have existed in France for thirty years without making any important progress; and there is no reason to doubt that it would have remained in its original haunts for thirty years longer, or, indeed, for an indefinite time, but for the changed condition of affairs which caused animals from the infected districts to be scattered over so many departments of the country. This changed condition was brought about by the wars of the great Napoleon, when it was not only necessary to drive herds of cattle to supply the armies from the infected mountain districts through provinces still free from the discases, but, to replace the losses occasioned by the introduction of the cattle-plague, the farmers were compelled themselves to draw upon the mountain provinces.

Even under these circumstances the only notable outbreaks which followed for the next twenty years were one in the vicinity of Montargis, about 50 miles south of Paris, and another in the district surrounding Valenciennes, in the department of Nord. In other words, during the half century preceding 1810 that pleuro-pneumonia had certainly been prevalent in Franche-Comté, it appears to have infected but five centers in France outside of this district. These centers were isolated; there was long distances between them, and at no time during the whole of this fifty years did the disease spread over the intervening country in the manner of an epizootic.

A period of ten years can still be added to this half century before there was any important extension of the infected district in France. This brings us down to 1820. Sixty years had elapsed since pleuro-pneumonia was known to exist in Franche-Comté, more than a century had passed away since the same disease had been clearly described in the adjoining country of Switzerland, and yet during all of that time by far the greater part of France remained entirely free from its ravages. In the few years subsequent to 1820, however, there was a remarkable extension of pleuro-pneumonia into many departments of France, where before it had been unknown. And this was not because the characters of pleuro-pneumonia had changed, but it resulted from an increased traffic and interchange of animals. In 1820 it had reached the Pyrenees.

In the infection of the province of Ardèche we have another striking illustration of the slow extension of pleuro-pneumonia in Europe. The adjoining district of Dauphiné had suffered from its ravages from time immemorial, but there being no interchange of animals, Ardèche was not infected until 1822. Even at this time the infection did not come as an extension of the disease from the neighboring Dauphiné, but it was brought by oxen from Franche-Comté, which were used to tow the boats on the river Rhône. There is another striking illustration of the characters of pleuro-pneumonia in this infection of Ardèche, to which I must call attention. The outbreak occurred in the winter; the cattle were nearly all in stables where they did not come in direct contact with each other; as a result the spread of the disease was limited, and it was soon entirely eradicated. This province enjoyed an exemption from the disease from this time until 1847, when a second introduction occurred.

Between 1820 and 1840, the disease extended over a large part of France, but it did not progress suddenly, nor sweep over a very great extent of new territory in any one year. Its extension was gradual; where it gained new territory it held it, and its spread was not by a rapid extension from farm to farm, but by the movements of cattle from one district to another, and by the mixing of sick and well cattle on the large herding grounds where animals belonging to many owners fed over the same pastures.

The infection of Belgium and Holland is another illustration of the slow extension of pleuro-pneumonia in Europe, when it was necessary for this extension to cross the boundary line of countries between which there was only a limited movement of cattle. Belgium was not infected until 1826, notwithstanding the fact that the disease had existed in Swabia within two hundred miles of the Belgian frontier for more than a hundred years. In 1827 it was particularly prevalent in Brussels, Mechlin, Louvain, and Diest, but it did not reach Hasselt, though this city is within twenty miles of Diest, until 1836. That is to say, this intervening twenty miles had preserved the cattle of Hasselt for nine years, and even at the end of this period they were not infected by a gradual extension of the malady from Diest, but by animals brought in from Flanders.

Substantially the same history is repeated in the infection of Germany, Austria, and Italy. It was no great distance from any of these countries to the districts which had been infected with pleuro-pneumonia for generations, and yet they remained free from the plague until about the year 1800, when the extensive military operations of France within their boundaries necessitated the transport of cattle for army supplies from the infected mountain herds to nearly every part of Europe.

There seems to be still certain restricted portions of Europe, where there has been little if any importation of cattle, to which the disease has not yet penetrated; for instance, it was stated at the International Veterinary Congress held in Brussels, in 1883, by M. Locusteano, that it did not exist in Roumania. (*Recueil de Médecine Vétérinaire*, 1883, p. 338.)

CONTESTS OVER THE CONTAGIOUSNESS OF PLEURO-PNEUMONIA IN OTHER PARTS OF THE WORLD.

America is not the only country in which the contagiousness of pleuro-pneumonia has been questioned. The insidious nature of the disease, its long period of incubation, the considerable proportion of animals which contract it in a form so mild as scarcely to be observed, and the number which are not at all affected by it, are characters which have always and in all countries been seized upon by those who opposed restrictive measures as evidence that this disease does not spread by contagion. In nearly all countries except our own the logic of events has in the course of time removed all doubts and has established beyond question the fact that pleuro-pneumonia in cattle is a contagious disease, and that although it may take its victims but one or two at a time it continues its ravages so long that in the end it proves one of the most destructive of all the animal plagues. Unfortunately it has usually taken so much time to convince people of this fact, and especially that part of the population who do not come in direct contact with diseased animals, that the disease has been given time to spread beyond control before any effective measures have been taken. The result is that even with a good veterinary service these countries are still suffering from the plague, and have up to this time found it impossible to eradicate it entirely. Great Britain, France, Belgium, Holland, Prussia, Austria, Australia, New Zealand, and South Africa are all examples of countries which might have been spared but for this fatal delay. Since we are now going through the initial period of this history in our own country, and have nearly or quite reached the crisis beyond which it will be impossible to rid ourselves of pleuro-pneumonia without a long and expensive struggle, it will prove a valuable and interesting study to compare the arguments urged against contagion in Europe with those advanced with the same object in the United States.

Notwithstanding the fact that Haller expounded the doctrine of contagion in 1773 with a clearness and force which can scarcely be improved upon at the present day, and notwithstanding his observation that even in Switzerland, the home of pleuro-pneumonia one might say, this disease does not arise except by contagion, there have been many to contest this conclusion since, even in the most enlightened countries of Europe.

Chabert considered the disease contagious, and the great influence of his name secured the general adoption of this opinion. Later Huzard investigated a disease of the lungs which prevailed among the milch-cows of Paris, and pronounced it a non-contagious disease. It would appear that Huzard's observations were made with cases of chronic pleuro-pneumonia, for from that time all acute inflammations were regarded as contagious, and all chronic forms, or those supposed to be chronic, were looked upon as non-contagious.

It was not long before it was learned by experience that this distinction would not hold good. Acute pleuro-pneumonia was not always fatal, nor did it always terminate by gangrene of the affected lung; on the other hand, it frequently passed into a chronic stage, and was followed by induration and the other results that had been supposed to be characteristic of the ordinary inflammations of the lungs. From this it was only a step to the conclusion that no form of pleuro-pneumonia was contagious. The idea of contagion had been reached by the observation of the progress of the disease in countries where it had long been known; the idea of its non-contagious nature was reached by a process of reasoning taking as a basis the character of the lesions which it produces. And this difference in the manner of studying the disease has caused the difference in the conclusions from that time to this. Those who studied the disease in the affected herds and saw it carried from stable to stable and from country to country with animals from these herds could plainly see that whatever the appearance of the animal might be when examined after death, the disease itself could be contracted by well animals coming in contact with sick ones. It was the old story of positive facts confronted by negative facts, and, owing to an equal value being given to each, the negative facts won the day, and the doctrine of the spontaneous origin and non-contagiousness of pleuro-pneumonia was quite generally accepted.

From 1800 to 1840, the opinion that pleuro-pneumonia was not a contagious disease seems to have steadily gained adherents, and it was during this long discussion that the disease was allowed to spread over all parts of Europe. The continued infection of new territory, however, and the more extensive scientific investigations from 1840 to 1855 brought evidence that was too strong to be resisted. Having admitted the contagiousness of the plague, the discussion then turned upon its spontaneous origin, and from 1855 almost to the present time there has been a large body of intelligent men in Europe who have insisted that contagious pleuro-pneumonia frequently originated from other causes than contagion—that it might be produced *de novo.* But the great advance which has taken place in our knowledge of contagion from the investigations of the last twenty years has led to the gradual abandonment of the theory that the contagion of this disease is produced *de novo*, and in the last International Veterinary Congress at Brussels there were but one or two delegates who advocated it. A resolution was passed unanimously, however, that practically the disease should be looked upon as arising only by contagion.

Returning to the arguments advanced against the contagiousness of this disease in Europe, I translate as follows from Mr. Delaford's treatise:

> In France, J. B. Huzard, Gaullet, d'Arboval, Didry, Taiche, and Gellé; in Italy, Lessona; in Germany, Dieterichs, Sick, Wagensfeld; in England, Fergusson, have advanced the opinion that this is not a contagious disease.
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> The greater part of the commendable authors whose names have just been mentioned have not supported their opinion by facts. In regard to this I repeat that science is no longer satisfied with opinions; it requires exact observations conscien-

ciously collected to carry conviction. I do not believe then that I should occupy myself with opinions; I will only attempt to examine published facts, and the conclusions which have been deduced from them to prove that pleuro-pneumonia is not a contagious disease.

A. LESSONA.—HIS IDEAS ON THE NATURE OF PLEURO-PNEUMONIA.

M. Lessona, professor at the veterinary school of Turin, published in 1836 a work of 206 pages in which he attempted to prove that the pleuro-pneumonia generally considered as contagious is only an ordinary sporadic pleuro-pneumonia with no power of transmitting itself. Reasons based on the cause, the nature, the seat, the treatment of pleuro-pneumonia, and finally facts of non-contagion were assigned for the opinion of our estimable and learned colleague. These will now be considered.

1. According to M. Lessona, pleuro-pneumonia is not a disease essentially gangrenous; it is due to constitutional causes, such as cooling of the surface of the body by damp winds, fogs, showers, snow, alternated with variable degrees of heat and cold, icy drinks introduced into the stomach, the fording of creeks and rivers of cold or icy water, warm, damp stables with collections of manure, respiration of impure air, &c., &c. These causes act on a considerable number of animals in different stables and pastures; and being felt throughout the same locality or in different localities they determine local outbreaks of pleuro-pneumonia which are enzootic or epizootic, but are not contagious, though they have this appearence and produce a belief in contagion.

2. As to the nature of pleuro-pneumonia, M. Lessona thinks that it cannot be contagious since this disease with mankind, the horse, the sheep, and the dog is not considered to be transmitted by contagion.

3. The contagious diseases of cattle in our climate, adds the author, are the charbonnous, typhoid, and gangrenous affections. Pleuro-pneumonia is not of the nature of charbon, since it is not accompanied with tumors, discolorations, or carbuncular infiltrations. The charbonnous diseases are developed spontaneously, and then are incontestably propagated by mediate and immediate contagion. Consequently, as nothing like this is seen in the course, termination, or extension of pleuro-pneumonia, this disease cannot be contagious.

The typhus is brought to us by foreign animals which come from Hungary; its advance is rapid, its progess is devastating, the contagion spreads itself in a short time over a vast extent of country. But these characters are not made apparent in pleuro-pneumonia; it is, therefore, not of the nature of the cattle plague, and cannot, consequently, like that disease, be transmitted by contagion.

It is an error to assert, adds M. Lessona, that pleuro-pneumonia is of a gangrenous nature. Gangrene can certainly be one of its terminations, but it is not the one which is most often observed, and even if the disease terminated by gangrene, I would still not admit the contagion, if contagion there is, except during the existence of this formidable termination.

These are the principal reasons with which M. Lessona combats the opinions of Brugnone, Toggia, Chabert, Laurin, Sandry, Leroy, and of our colleague and friend M. Lecoq, in the analysis which he makes of their works on the nature of pleuro-pneumonia, and with the object of certainly demonstrating the non-contagiousness of this disease. I will add here that these reasons are also used by the French, German, Belgian, and English anti-contagionists.

I reasoned in this way myself ten years ago, and I said, in common with a number of veterinarians whose knowledge, experience, and talents of observation are well known, "I will believe in the contagion of the pleuro-pneumonia when I have positively observed it." To-day the facts which have come before me have entirely convinced me to the contrary, and cause me to say that this disease is contagious.

I am entirely in accord with our Italian colleague in regard to the nature of the disease. Certainly pleuro-pneumonia is neither a charbonnous, typhoid, or gangrenous affection; it is an inflammation of the lungs and pleuræ, the ordinary results of which are pleural effusion, and induration of the pulmonary tissue. But can we say because this is its nature that the disease is not endowed with the pernicious property of transmitting itself by contagion? I do not think so. Are hydrophobia, sheep-pox, glanders, foot-rot of sheep, scabies, &c., charbonnous, typhoid, or gangrenous diseases? And nevertheless these diseases are assuredly contagious. In the same manner that these give birth to a special virus, may not also the pulmonary alteration which constitutes pleuro-pneumonia produce a volatile, reproducing principle which, carried from the diseased lung with the expired air, is susceptible of being introduced by the respiration into the lungs of animals in good health, and of communicating to these organs a disease of the same nature as that which gives birth to it? And besides, it is not by comparing a disease to some other disease that we reach a solution of such a question, but by well-observed and decided facts. Without doubt in many cases pleuro-pneumonia may develop spontaneously by the influence of local causes, and particularly such as those that M. Lessona assigns to it, and that I admit with him; but why cannot this disease after once developing reproduce itself by contagion? Are not rabies, charbon, scabies, acute glanders, &c., diseases which develop spontaneously under the influence of local causes, and which afterwards are communicated by contagion? Then why cannot it be the same in regard to pleuro-pneumonia? Why not admit that local causes produce it and that it is then communicated by contagion?

If pleuro-pneumonia is contagious, says M. Lessona, it should affect a large number of animals at a time, be propagated at a distance, and all animals exposed should contract it. But, says our colleague, this is far from being the case, for pleuro-pneumonia is generally a local disease. Without doubt the disease under consideration is not propagated in the same manner as typhus or charbonnous fever, and Brugnone, Chabert, and Toggia have certainly exaggerated this contagion; but because pleuro-pneumonia is not communicated to all the animals which are exposed to its influence, because it does not extend itself over a vast territory, should this difference authorize the conclusion that it is not contagious? Are all virulent diseases transmitted in the same manner? Does not glanders differ in this respect from charbon, charbon from typhus, and rabies from mange? Then why should we deny a peculiarity in the transmission of pleuro-pneumonia when we admit peculiarities with other contagious diseases the nature and seat of which are different?

Some persons assert that they have seen cattle in fine condition standing in the same stable and eating from the same manger with animals affected with pleuro-pneumonia and not contract this disease.

I do not contest these facts, for I have seen them; but can we say because of this that the disease is not contagious? Certainly not. It would be extremely unfortunate if people or animals should contract a disease every time they are exposed to its contagion.

Assuredly if we reflect that, with pleuro-pneumonia as with all other contagious diseases, the transmission requires for its occurrence the presence of a virus, the integrity of this virus, its deposit on living tissues, its absorption, and a susceptibility of the subject to contract the disease, and that the absence of any one of these five conditions neutralizes the contagion, it is easily conceived how and why animals may not contract a contagious disease, though exposed to others which are affected with it.

As to saying that because no fact proves that the pleuro-pneumonia of man, the horse, or the sheep, is transmitted by contagion, it should be the same with the pleuro-pneumonia of cattle, this assertion appears to me entirely gratuitous. In veterinary pathology analogies should only be invoked with the greatest circumspection, for the species of animal, its constitution, its temperament, the intimate organization of the part attacked, have a singular influence on the form and the nature of diseases, even though they apparently develop under the influence of the same causes, resemble each each other in many of their symptoms, and affect the same organs.

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M. Lessona not only gives the reasons which have been considered above, but he supports them with instances of non-contagion which he has observed and which have been communicated to him.

These cases are twenty-two in number; three were observed by the author, and the remainder were related to him by the veterinarians Luciano, Ferrari, Robecchi, and Perrotti. I will give a succinct but accurate account of them and afterwards examine their value.

Of the fifteen facts given to Lessona by Luciano, twelve prove nothing in favor of the non-contagiousness of pleuro-pneumonia. The substance of them is as follows: The disease occurred in different herds of cattle, composed of 10, 30, and even 100 beasts; of this number 2, 4, 6, 10, 20, 30 animals were affected by it and died or were cured; the remainder were not sick. Now, because all the animals in these herds were not affected with pleuro-pneumonia, Luciano and Lessona conclude that this disease is not contagious, because if it were it should have been communicated to the whole herd attacked and even to neighboring herds.

Can we prove in this way that a disease is not contagious? Must all the animals exposed to a contagion necessarily contract the disease before we can admit a contagion? But there is no contagious disease, either with man or animals, no matter how subtle it may be, with which this occurs.

In the three other facts reported by M. Luciano, pleuro-pneumonia occurred in stables, and was not communicated to the other cattle on the same farm. According to the author, who gives no details with his observations, the disease should have been communicated if it had been contagious. These facts prove absolutely nothing to me in regard to the non-contagiousness of the disease, for it is not communicated at a great distance as is charbonnous fever, sheep-pox, or typhus, and it is sufficient to isolate the animals in stables to prevent the extension of the contagion in many cases even to other stables which are near those that are infected. I have observed such facts; others have also seen them, but I have not been able to accept them as a proof of the non-contagious character of the disease.

Three observations were communicated to M. Lessona by Ferrari, Robecchi, and Parrotti, and were inserted by him in the journal called *Le Propagateur* (*Propagatore*). All of these, like those given by Luciano, are wanting in those circumstantial details which bring conviction to the mind of the reader. Then, as pleuro-pneumonia in each case developed in a large herd, but only attacked ten or fifteen animals, Lessona concludes that it is due to constitutional causes. Still, even these causes should act alike on all the animals which are exposed to them; why then did the disease confine itself to so small a number of them? Could M. Lessona give a satisfactory explanation of this fact? These observations have consequently no value for me.

The facts reported by our Italian colleague, M. Lessona, number four. The first is as follows: In the month of November, 1827, pleuro-pneumonia broke out in a herd composed of thirty-eight animals after the return of this herd from the Alps of *Giavène* and in the locality called *Margaria di Collegno*. Eight animals died out of twelve attacked by pleuro-pneumonia. The cows of this herd had all been raised by the proprietor, with the exception of two animals purchased in the market of Susa. One of the latter sickened first, then all of the others successively. The whole herd had suffered with bad weather in the mountains, and according to M. Lessona it was that which caused the pleuro-pneumonia among the animals composing it.

I regard this observation as very incomplete and proving nothing in favor of non-contagion. In the first place, did not the two strange animals come from an infected stable, as so often happens with cows purchased in the markets? M. Lessona is silent in regard to this. Was this the only herd affected with the disease? Were the animals of neighboring proprietors, which had doubtless been equally subjected to atmospheric inclemencies, also sickened? Lessona does not trouble himself about these facts, or, at least, he says nothing in regard to the subject.

The second observation, made on several herds grazing in the Alps and among the animals of which pleuro-pneumonia manifested itself without being communicated to other herds of the vicinity which pastured on the same grounds, is wanting in details, and cannot be considered as conclusive.

Mr. Lessona has been more precise in regard to the following fact:

"M. Berthoglio, of the farm called *Gros Cheval*, owned a herd composed of 28 cows. Of these 3 cows had passed the winter in the stables of the farm, and the 25 others had been purchased in April and May at the markets for animals in the vicinity. Of the 3 cows which M. Berthoglio had owned and which now made part of the herd, 1 died from the effects of parturition, and the 2 others were attacked with pleuro-pneumonia the 8th of June and perished.

"The 26th of the same month the 25 remaining cows were sent to pasture in the Alps, this being 16 days before the ordinary time when cattle are taken to pasture on the mountains. To this herd were added 7 head of cattle belonging to other proprietors of the *commune*.

"Of the 25 cattle purchased by M. Berthoglio 14 were attacked with pleuro-pneumonia between the 10th of June and the 19th of September; 5 died, and 9 were cured. The 7 cows which belonged to various owners were not sick."

M. Lessona concludes from this observation that pleuro-pneumonia was not transmitted in this herd by contagion because: (1) The disease did not show itself in the first place among the cows purchased by M. Berthoglio, but it attacked the two that he had owned and kept in his own stables. (2) This disease was not contracted by the 7 animals of neighboring owners which grazed on the same pastures. (3) The people who visited the sick animals of M. Berthoglio did not carry the disease to their herds. (4) Later, after the return from the Alps, no animal became sick. (5) The causes which produced the disease were on the one hand the low, damp, and warm stable in which the animals were lodged, and on the other hand the exposure to cold, from the animals having been conducted to the mountains 15 days before the ordinary time.

I would remark here:

1. That in accord with Lessona I admit that the heat of the stables may have been sufficient to give rise to pleuro-pneumonia among the cows of M. Berthoglio; I admit also that the extremes of temperature may have given rise to it in the mountains. But could we not also argue that the animals which were placed in the stable where the first cows were sick were infected there, and that the disease did not develop with them until after reaching the mountains? I am the more persuaded to believe that this was the case since two days had scarcely passed after the departure of the herd for the mountains (June 10) when the third cow of M. Berthoglio was attacked with pleuro-pneumonia; then counting from this day all the others were successively attacked with this disease, in June, July, August, and September. Now is a pleuro-pneumonia due to atmospheric influences which persists during all the finest season of the year in the mountains and which remains four months in a herd? And is it

not reasonable to suppose that the cause which determined the disease in the mountains was not that assigned by M. Lessona, but was really contagion?

2. As to saying that pleuro-pneumonia should have been communicated to the animals belonging to M. Berthoglio's neighbors, I will repeat that in a herd affected by the disease under consideration there are always animals which do not contract it, though exposed to its contagion. Besides, it is very rarely that pleuro-pneumonia attacks all the animals in a herd.

To recapitulate: I believe that the observation of M. Lessona may be strongly contested as proving the non-contagiousness of pleuro-pneumonia. What fortifies me in this position is that M. Lessona was not certain himself of its non-contagiousness; for when consulted by the health magistrate of the commune he stated that the herd should be brought from the Alps and placed upon an isolated farm, and that if other animals were attacked these should also be sequestered. But why does M. Lessona prescribe these means of isolation if he believes that the disease is not susceptible of being transmitted by contagion? Certainly this would be a superfluous sanitary measure and contrary to the interests of M. Berthoglio.

The fourth observation is as follows: "In the month of August, 1832, pleuro-pneumonia manifested itself," says M. Lessona, "among the animals of the farm of *La Motta*, which was managed by two farmers, M. Balbis and M. Macagno.

"M. Balbis owned 25 head of cattle. Of this number 4 were attacked, 2 between August 1 and 22, and 2 between October 1 and 10; 3 of these recovered and 1 died."

M. Lessona attributes the development of the disease among the 25 animals which had been owned by M. Balbis for a year to local causes. "Besides," says he, "if pleuro-pneumonia had been contagious it would not have been confined to only 4 beasts among the 25 comprising the herd of the farm."

I am disposed to believe with M. Lessona that the pleuro-pneumonia under consideration was determined by local causes, but I do not share his opinion as to its non-contagious nature, because it did not attack a greater number of animals in the herd.

With M. Macagno, a neighbor of M. Balbis, pleuro-pneumonia attacked the animals about October 15; consequently, as I will remark, it was at the time that the disease still existed among the animals of M. Balbis. Macagno owned 18 head of cattle; 3 were attacked, of which 2 died and 1 recovered. At the time these animals were sick M. Costanzo brought to the farm 4 cows that he had owned more than a year, and 2 of these were soon attacked with pleuro-pneumonia, of which one died and the other recovered.

According to M. Lessona it was not contagion which transmitted the disease from the animals of M. Balbis to those of M. Macagno, nor from the latter to the 2 cows of M. Costanzo which had been introduced into the infected stables, because: (1) In Italy no contagious diseases are known, except carbuncular affections, and, as the pleuro-pneumonia under consideration was not of a charbonnous nature, it could not be contagious. (2) The disease was not constantly fatal. (3) The real cause of the disease was feeding too early in the morning or too late in the evening on fresh and moist pasture, and falling in the river where the animals drank. As the very marked instance of contagion to the cows of M. Costanzo, M. Lessona attributes it to the bad condition of these animals and their exposure to the causes just mentioned.

One must certainly be a strong anti-contagionist to be satisfied with the facts reported by M. Lessona. As for me, I find them very incomplete, and I believe they cannot strengthen the opinion of those veterinarians who believe in non-contagion. Our colleague, M. Gellé, was, however, satisfied to accept these facts as a basis for his opinion, and he hastened to follow M. Lessona in writing that pleuro pneumonia is not contagious. Nevertheless our estimable colleague, M. Lecoq, had reported facts of contagion which M. Lessona had vainly sought to controvert; but M. Gellé took no account of these facts, nor of many others which had been published and were known. He disposed of the question by saying he could not recognize contagious pleuro-pneumonia in the description that had been given of it by his honorable colleague of the Lyons school. For my part I am certain that M. Lecoq has studied pleuro-pneumonia better than M. Gellé, because he has seen sick animals affected with it, while our colleague of Toulouse appears to have known the disease only from the books that he has compiled.

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Here are some other facts which appear more conclusive than those of M. Lessona. They are attempts to produce contagion by stabling healthy animals by the side of cows affected with pleuro-pneumonia. * * * I will relate these attempts, which have all been without result:

"I desired," says Dieterichs, "to convince myself if the emanations which escape from animals affected with pleuro-pneumonia can transmit this disease to healthy beasts placed by the side of them. For this purpose I made use of a cow five years old, after having assured myself of her perfect health by observing her when eating, when drinking, and when made to cough by a slight pressure of the larynx. I placed her between two oxen sick with pleuro-pneumonia, in a very narrow stable where there was scarcely space for three animals. All were fastened with long halters in order that they might reach each other's forage. The oxen ate very little, since the disease was already in an advanced stage; the cow, on the contrary, not only ate the small quantity of food that was placed before her, but she consumed as much as possible of that which had been soiled by the nasal mucus and saliva of the oxen. This cow was left there for only two days and was then returned to the stable and stall from which she had been brought. She was replaced by another cow also very healthy. The latter remained with the two oxen for a day and a half, when one of them died. She remained two days and a half longer with the other one, at the end of which time it was killed and showed the existence of a perfectly characteristic pleuro-pneumonia. The result of this experiment," says Dieterichs, "demonstrated to me the non-contagiousness of the disease." (Extract from the work of Dieterichs, published in 1821.)

Veith, director and professor at the veterinary institute at Vienna, thus expresses himself in his treatise on the epizootics of the principal domestic animals, published in 1831, article epizootic pleuro-pneumonia:

"In the majority of cases this disease is not of a contagious nature. In numerous experiments that we have made, by putting in immediate contact healthy animals and those infected, contagion has not occurred, even when the healthy animals licked the mucus and purulent secretions which dropped in abundance from the nostrils and mouths of the sick ones. Note, that the healthy animals had been brought from a distance and from localities which had been preserved from the disease."

The following is an experiment attempted in France by the old practitioner Gaullet:

"I had been commissioned," says Gaullet, "to proceed to the commune of Bligny, where pleuro-pneumonia existed among the cattle. There had already been 15 animals, consisting of oxen, cows, heifers, and young calves, carried off by the disease, and 10 others attacked. I caused the sick animals to be separated from the healthy ones, though I did not judge this disease to be contagious. To render my opinion more convincing I attempted an experiment on my own account and risk. I purchased a cow in good condition and six years old, from a neighboring commune where the disease was not known, and had it brought to Bligny and put in a stable with a cow and an ox affected with the epizootic and at a stage that was judged to be incurable. The cow was placed between these two animals. The ox died three days after the beginning of the experiment, and the sick cow lived but five days. At the autopsy of the ox I took serious and purulent liquids still warm, and after making a vertical incision 2¼ inches in length between the ribs and loosening the skin surrounding it, introduced a pledget soaked in these liquids and maintained it in place with a bandage. The animal was not allowed solid food for two days, and during this time a considerable swelling showed itself around the inoculation wound, which was accompanied with great sensitiveness; a few days later an eschar formed and in a short time was detached. The wound was dressed with *digestif*, and the swelling soon diminished, the wound becoming cicatrized within fifteen days.

"This animal was not even affected with an indisposition. I sold her to a man who kept her for two years in good condition and health, but, as she was a small milker, he sold her to a butcher for slaughter. Being present when she was opened, the organs of the chest were found perfectly healthy."

To these facts we might oppose the similar attempts made by Hertwig, who succeeded in causing the transmission of pleuro-pneumonia to three healthy cows which had been placed by the side of sick ones, and which died of the same disease, and also the numerous facts of contagion which I have reported from infected stables into which perfectly healthy cattle had been introduced.

For me these experiments only prove that in some cases healthy animals placed by the side of sick ones resist the contagion with which they are menaced. Similar facts have always been observed during the existence of all contagious diseases, whatever their nature.

The above extracts are sufficient to show how the nature of pleuro-pneumonia was misunderstood and its contagious character contested on the Continent of Europe, even by the members of the veterinary profession. Even Delafond was willing to admit that this disease, and also glanders, rabies, and other contagious fevers, frequently originated spontaneously. The disastrous experience of the present century has removed all doubts of the contagiousness of pleuro-pneumonia in Europe, and the rapid advance of scientific knowledge is making it equally clear that the belief in its spontaneous development must also be abandoned. I have referred at such length to the arguments and facts which were used in Europe because they are of peculiar interest at this time, since exactly the same line of reasoning and the same characters of the disease are now being used to demonstrate the non-contagiousness of the same disease among our own cattle. And incidentally these arguments show very conclusively that the pleuro-pneumonia of Europe has exactly the same characters as the pleuro-pneumonia of America.

It is so very interesting and important to note how the people of different countries who are unacquainted with the nature and history of this disease are always ready to seize upon the same peculiarities of its course in an attempt to show how it cannot be dangerous that I will make some brief allusions to its history in England.

Pleuro-pneumonia was carried by cattle from Holland to Ireland in 1839, and was found to have reached England in 1842. As late as 1859 we find an article published in the Transactions of the Highland and Agricultural Society of Scotland which was written by Professor Dick, principal of the Edinburg Veterinary College, to demonstrate the non-contagious nature of epizootic diseases. Like the continental veterinarians referred to above, he produces a number of instances to show that pleuro-pneumonia is produced by atmospheric causes, but not by contagion. Where it occurred in stables it was attributed to too much or too little ventilation, and when it was found among cattle on pastures he thought it was produced by exposure to atmospheric changes. He was consistent in his views, however, so far as to deny contagion with all epizootic diseases, and he contested the contagiousness of the cattle plague (rinderpest) as strongly as that of pleuro-pneumonia. It is said that he even refused to admit that hydrophobia could be transmitted by the bite of a rabid animal.

On the subject of pleuro-pneumonia in England, Dr. George Armatage writes as follows:

> From an ignorance of the nature of epizootic diseases many authorities were inclined to regard pleuro-pneumonia, as now seen, as merely an extension of the sporadic form with which they were already acquainted (Youatt, to wit). The contagiousness was imperfectly made out, and frequently denied, and circumstances favorable towards establishing proof were commonly overlooked. * * * Many persons also argued that the disease would die out in a year or two, basing their assurances upon the observed fact in other complaints, viz., the tendency to become milder after a time. The history, however, of pleuro-pneumonia epizootica, as it has pursued its ravages since 1842, tells a different story.
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> The subject of contagious cattle diseases has not yet received that careful consideration, with the view to their prevention and eradication, which they deserve. It is only now at the time we write that any widespread feeling exists or desire is evinced towards that end. Hitherto newspaper editors and paragraph writers, who knew nothing of the question, blindly wrote against the probability of their being of foreign origin, and refused admittance to their columns of any letter or expression which was calculated to rouse the public to a sense of the approaching danger. Even the direful tale told, in unmistakable words, by the cattle plague of 1865 and 1866 had made little impression, and to the tone of the press particularly is due much of the want of organization and slow progress which has marked the efforts to annihilate the scourges that now threaten to destroy our flocks and herds. * * *
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> As in all epizootic affections among cattle, we trace the origin of *pleuro-pneumonia epizootica* to contagion propagated by the continued and indiscriminate movement of animals to and from fairs, markets, &c. The mystery of the origin of the first case proves as great and insurmountable here as in the allied diseases of man, yellow fever, cholera, typhus, &c., and among animals, rinderpest, small-pox, and hydrophobia. That there are centers of origin we have no doubt. As man falls a victim to cholera in the Pontine marshes, or yellow fever at New Orleans, as soon as he arrives, in like manner healthy cattle taken from places perfectly free from diseases would become a prey to the indigenous epizootics which prevail in the steppes of Eastern Europe and borders of Asia, &c., with mortality proportionately excessive, if they were collected and imported from those countries. We, as well as cattle, suffer to an aggravated extent because infected subjects are allowed to go about indiscriminately, and scatter the poison from their bodies wherever they go.

> That the prevalence of pleuro-pneumonia is alone due to contagion is satisfactorily proved by several circumstances. *First.* Admitting for a moment that all contagious diseases arise spontaneously where filth abounds and neglect of sanitary laws exist *ad libitum*, how does it happen that during a whole century this affection was not seen, and particularly as cleanliness and ventilation were not so much regarded as now? *Second.* How does it also happen that pleuro-pneumonia epizootica has never been witnessed in certain isolated districts, where cattle are not taken from other parts, but bred upon the spot, yet filth and neglect of sanitary regulations is as certain in many of them as the buildings upon the farms? *Third.* Why are the dairymen and others who frequently resort to fairs and markets to replenish their stock always the greatest sufferers, even where every caution is exercised and attention paid to sanitary regulations?
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> The answers are simple and irrefragable. Although filth is an acknowledged cause of disease in man and animals, its baneful effects are manifested differently upon each, in accordance with climate, latitude, and other conditions. These peculiarities constitute different tracts of country as the home of certain maladies, and, in addition, we need other conditions, but which are not well understood, to generate epizootic or contagious diseases. Beyond such localities they can never arise spontaneously. Although in our own country filth may abound, we never hear of it generating the yellow fever of New Orleans; and wherever we find pleuro-pneumonia epizootica, rinderpest, small-pox, or hydrophobia, there we may, without doubt, trace their origin to the effects of contagion only. If the causes of rinderpest or pleuro-pneumonia and aphtha were in reality present within the limits of our kingdom surely the experiences of our unusually dry and hot summers, with deficient water and stagnant pools and accumulations of filth and putrescence beneath our very noses, would have developed them in accordance; and likewise the severe winters, so destructive to the progress of putrefaction, would have caused them to disappear in proportion. But such has not been our experience. These affections are not influenced by heat, drought, or cold; they maintain a steady, certain, and uncontrollably destructive influence under *all* conditions of temperature, and appear to be aggravated only by fresh introduction of virus among sound, healthy stock. (Trans. Highland and Agricultural Society of Scotland, vol. 3, fourth series, pp. 51–53; vol. 5, fourth series, p. 198.)

Notwithstanding the history of pleuro-pneumonia in England, and the conclusion finally reached to the satisfaction of all intelligent persons that the disease was propagated by contagion alone; notwithstanding the infection of South Africa in 1854, and the enormous losses caused by its dissemination over the vast grazing grounds of that country—when pleuro-pneumonia was introduced into Australia in 1858 there was the same contest over its contagiousness and the same opposition to suppressive measures that had marked the history of this disease in its progress over the various nations of the Continent and over the British Isles. A pleuro-pneumonia commission was established to investigate the contagiousness of the disease.

> They were to a man all non-contagionists, from Ralph to Rowe and M'Coy, and they fetched healthy cows from Tasmania, where pleuro had never been, placed them in stalls beside diseased animals—got there in abundance—inoculated them in various crucial ways, and then declared their inability to communicate the fever by contagion, reported the result to the legislature, and based upon their failure an advice deprecating any further legislative interference or alteration of their veterinary police; and yet the Government printer's ink was hardly dry upon their notable report, when a counter report came from the butchers of Geelong, who had bought for slaughter the experimental bullocks, that the animals were all diseased, unfit for human food, and demanding back their money. (Veterinarian, September, 1875, p. 681.)

It seems remarkable that such skepticism in regard to the contagiousness of the disease should exist in Australia at so late a date and after the experience of so many other countries, but history appears to repeat itself, with almost unvarying certainty, in every new country into which this disease gains access. Extending at first slowly and almost imperceptibly, as it must necessarily do, owing to its peculiar character, its danger and contagious nature are doubted, and while this uncertainty prevents action the plague gradually makes headway until it reaches the cattle markets of the large cities and enters the channels of commerce, when it is disseminated with amazing rapidity, and before people have awakened from their dream of security it is practically beyond control.

This history came very nearly being repeated in Massachusetts during the six years subsequent to May, 1859, in which pleuro-pneumonia existed in that State. The public apathy, the doubts in regard to the nature of the disease, the demand for temporizing measures and experimentation, were obstacles in the way of its suppression there as they had been in France, Germany, Holland, Italy, England, and Australia, but fortunately there were a few intelligent men who understood the situation, and who had the determination and influence to secure and enforce those regulations which alone have been successful in stamping out this disease. The disease was clearly traced to four animals imported by Mr. Chenery, of Belmont, from Holland, and the commissioners plainly stated in their memorial to the legislature that although the cattle of 75 owners had been discovered infected the origin in every case could be traced directly or indirectly to this particular herd. (Report Massachusetts Board of Agriculture, 1860, p. 6–10.)

In spite of this clear history of the introduction and dissemination of the disease, and notwithstanding the most incontestable instances of contagion presented in very forcible language by the commissioners appointed under a special act of the legislature, there were still many who contested the diagnosis of the disease, and who doubted its contagiousness. Introduced in May, 1859, it was allowed to spread unchecked for nearly a year, and then it was necessary to slaughter 932 animals to stamp it out. This heroic treatment extirpated the disease completely in the localities where it was practiced, but after more than a year had elapsed it was found to exist in other places to which it had been carried by animals, infected in the first outbreak, which had eluded the investigations made at that time.

The committee appointed by the Board of Agriculture to investigate this second outbreak closed their report as follows:

> There still being doubters in the community as to the existence of contagious pleuro-pneumonia, earnest attention is called to the thorough and convincing report of the first board of commissioners, with accompanying documents, in the Report of the Secretary of the Board of Agriculture for 1860.
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> We believe that no person, however prejudiced he may have been, who has been present at the examination of affected animals, has failed to become convinced of the contagiousness of the disease, and it would seem impossible that any one can doubt this fact who will examine the various reports that have been made upon it in Europe and in this country. (*Op. cit.* 1861, p. 134.)

The objections to legislative action and the doubts in regard to the nature of the disease appear to have increased rather than to have diminished, however, for in the Report of the Secretary of the Board of Agriculture for 1862 we find a report of the same committee submitted on the 15th of January, 1863, in which the following statements occur:

> The committee now further report that they were diligent and untiring in their exertions, by the presentation of facts which were daily transpiring, to urge upon the legislature the importance of immediate efficient action.
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> After much delay a bill was passed authorizing the governor, with the consent of the council, to appoint a board of three commissioners whenever in his judgment the public good required.
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> This measure was strongly opposed at every stage in both branches of the legislature, and after having become a law the council were slow to be convinced or to allow that the public good required any such appointment, and would consent to make one only by appointing a board the majority of whom were opposed to any legislation on the subject, and who were known disbelievers in the *contagiousness*, if not in the existence, of any such disease, and we believe it is no injustice to the commissioners

to say, that at the time of their appointment none were more thoroughly opposed to the common theory of the disease, and the past action of the State, and former board of commissioners, than were two of the gentlemen composing the present board.

In an address to the farmers of Massachusetts these commissioners use the following language:

> Two of the three commissioners commenced their labors with a feeling that by careful inquiry, and by thorough examination, they should be enabled to demonstrate to the public the inexpediency of the action of the former board of commissioners as well as the groundlessness of the apprehensions of many in regard to the fatal character of the disease.
>
> A number of persons had published treatises to prove that pleuro-pneumonia was generated in poorly ventilated barns, and was not infectious. The facts, as developed to the commissioners, have constrained them to discard their first impressions, and to deny the positions of the various writers before alluded to. They have, moreover, thoroughly convinced themselves that the worst apprehensions in regard to the disease are well founded and wise. They have found the disease prevailing in barns of every variety of structure and of all degrees of ventilation, and even in the open fields. They have traced it from root to branches, whither it flows as surely as the sap flows in trees. They do not find a single case outside of the line of transmission. As surely, as every rivulet tends toward the sea, does each case connect itself with its fountain head. The conclusion is irresistible, that if any disease be infectious this one is. (Rep. Mass. Board of Agriculture, 1862, p. 20.)

In the report for 1863, we learn that the board of commissioners had resigned because the legislature refused an appropriation that would make their labors of any avail. The governor had directed that experiments be made to test the contagiousness of the disease, and the result of these, as is so frequently the case where scientific methods are not used, was very unsatisfactory. The situation at this time is an interesting example of the difficulties and uncertainties connected with the control of such diseases by State authorities. The secretary of the Board of Agriculture commenced his report for 1863, as follows:

> There seems to be now a reasonable certainty that we shall leave that monstrous curse to the farming interest, commonly called pleuro-pneumonia, as a legacy to posterity, not because it is out of our power to extirpate it, but because we are unwilling to lift a hand with sufficient energy to put it down. The old adage that *delays are dangerous* was never more fully illustrated than it has been in the history of the efforts made from time to time, spasmodic in their character, to get rid of this disease.
>
> Early in March, 1860, it was announced to the legislature that pleuro-pneumonia had broken out in North Brookfield, carried there by cattle bought in Belmont, near Boston, and that it was spreading from herd to herd. The legislature were also promptly informed that a small appropriation would check and exterminate it there. Now, if action had been taken without delay a very small sum would have effected the object. That is easily and clearly demonstrated by events which subsequently took place.
>
> But a delay of thirty-five days followed, and in the meantime every day and every hour was spreading the contagion, not only by buying and selling but by the working of cattle in the highways, bringing them necessarily into constant contact, the sound with the infected, so that before the first able commission was really created and ready for duty under the act approved April 4, a very large number of animals had been exposed and many had become diseased. The danger had increased day by day in geometrical ratio, and the sum appropriated, *which, if granted when asked for, would have been amply sufficient*, was found to be wholly inadequate to effect the object.
>
> As it was, so thorough and efficient was the commission, the disease was extirpated in that section. But a single yoke of oxen had escaped and could not be traced. Their history is not generally known to this day.

From the report of 1864, we learn that the board of commissioners had been reorganized, experiments were still in progress to test the contagiousness and curability of the disease, and one of the commissioners submitted a minority report in which he attempted to throw discredit upon all that had been done, and to show the uselessness of slaughtering the diseased cattle.

In the report for 1865–'66 it is stated that the labors of the commissioners had been so far successful that but few cases of pleuro-pneumonia had occurred during the year. The experiments seem to have given satisfaction, it being reported that of six animals exposed four had certainly contracted the disease (pp. 6–22).

It was not until December, 1866, that the commissioners made their final report, congratulating the people of the commonwealth on the extinction of the disease. The last cases, however, had occurred in October, 1865. During the six years the commissioners had killed 1,164 herd of cattle, and a number of others had been slaughtered by the selectmen of the various towns. (Report 1866–'67, pp. 6–10). From that time until the present no cases of pleuro-pneumonia have been discovered in the State of Massachusetts.

I have referred at this length to the difficulties encountered by the commissioners in Massachusetts, the objections which were raised to their conclusions, and the reflections cast upon their motives, because this outbreak has become a matter of history and the wisdom of their action is no longer questioned. It is a history which has been repeated in every country, I believe, to which pleuro-pneumonia has been introduced; but there was this difference: in Massachusetts intelligent counsels finally prevailed, and the disease was exterminated, while in most other places those who doubted its existence and contagiousness, and who demanded months or years of experimentation, have succeeded in delaying efficient legislation until the contagion was spread beyond the possibility of extermination. There were the same objections raised to State action in New York in 1879, and these were finally sufficient to arrest the work before the disease could be exterminated. And now, when the same disease has been carried beyond the Alleghanies, when it has penetrated into the great cattle-raising States of the Mississippi Valley, a new set of inexperienced men, who are ignorant of the history of pleuro-pneumonia in America or in Europe, are raising the same questions, and bringing forward the same line of argument which delayed action in New York and Massachusetts, and which brought devastation and destruction to the herds of France, Italy, Germany, Holland, England, South Africa, and Australia. It remains to be seen whether the people of the United States, the most intelligent and progressive of any in the world, will learn wisdom from the experience of other countries, or whether history and science will both be discarded and our cattle left unprotected from this destructive plague. What a comment for the future historian to make, that the United States of America in 1884 had no more intelligent policy to enforce in regard to the contagious pleuro-pneumonia of cattle than had the ignorant peasantry of the French and Swiss Alps in the early part of the eighteenth century.

INSIDIOUS CHARACTER AND VAGARIES OF PLEURO-PNEUMONIA.

The insidious nature of pleuro-pneumonia has been insisted upon by most writers who have treated of this disease. The long time which elapses between exposure to the contagion and the appearance of the first symptoms increases the difficulty of tracing infection to its source; and the fact that some animals which have had a light attack and are apparently sound are still able to infect others, and that some do not scatter the contagion even when very sick, have been used in all countries to discredit the transmission of the disease by contagion. That these characters have been observed in Europe as well as in America is apparent from the extracts already made from European literature.

A few more references bearing directly upon the subject are given below, to remove all chances for misapprehension. Dr. Armatage writes:

> From the lengthened period which elapses between the occasion of known contact with diseased animals and appearance of first symptoms, together with the lingering or tardy progress of diseased action, it is known as *subtle* and *insidious.* Whatever may be the subtle and deceitful nature of the plague (rinderpest), pleuro-pneumonia epizootica possesses those powers in a thousand-fold more fatal degree. The seeds of contagion lurk among stock for weeks or even months, and eventually break out in isolated and often not well-marked symptoms, and with the death of the first victim proprietors are sometimes induced to consider that the disease has altogether departed again. However, after a few more weeks another victim falls, and, in the lapse of time of more or less duration, another and another, and so on until all are taken.
>
> It may be, also, that the owner, ignorant of what he has to contend with, purchases new stock, one by one of which goes in the same manner, and naught but absolute ruin stares him in the face. The insidious nature of the affection was the cause of its introduction to Australia and America, and has alike, in many cases, caused its extensive propagation in our own country long before proprietors were acquainted with its presence. (Transactions of the Highland and Agricultural Society of Scotland, No. 5, 4th series, p. 54.)

Professor Walley, in his excellent work entitled "The Four Bovine Scourges" (pp. 15–17), writes as follows of the "vagaries" in the spread of contagious pleuro-pneumonia:

> These are very numerous, so much so, in fact, as to have led many observers to assert that the disease is neither infectious nor contagious.
>
> But these very vagaries tend rather to prove than to disprove its infectious nature, for it is a fundamental principle of the spread of zymotic diseases that vagaries in their propagation arise; and this is due to the well-known laws which govern all of them. These are:
>
> *Firstly.* That the poison shall be of sufficient potency and vitality to reproduce itself when brought into contact with favorable pabula.
>
> *Secondly.* That the system of an animal shall be capable of receiving it.
>
> *Thirdly.* That once having received the poison, it shall be capable of providing it with a suitable pabulum and a sufficient amount of nourishment to enable it to grow and multiply.
>
> *Fourthly.* That the means of conveying the poison to healthy animals shall be forthcoming.
>
> 1. It very often happens that the poison is of such a mild character, or has been so deprived of its virulent properties—by dilution, or from the system of its host not furnishing it with suitable nourishment—as to do away with its subsequent power of reproduction. Dilution takes place also in traveling, by admixture with atmospheric air. Its potency is also reduced by exposure to disinfectants, frost and other forces inimical to its well being.
>
> 2. The systems of nearly all animals are quite proof against certain poisons; this is notably the case with the disease under consideration, as it can scarcely be propagated to any other animal than the ox, no more than variola ovina can be produced in any other animal than the sheep.
>
> 3. Although an animal may have received a poison into its system, and though it may have found the particular pabulum in which it is usually reproduced—as the liver, lungs, intestines, &c.—circumstances may have been previously in operation which shall have led to the entire removal from the system, or at least its complete alteration, of the material upon which the particular poison usually feeds and multiplies; thus medicines—catalytic or purgative—setons, rowels, vaccination, inoculation, or the pre-existence of another zymotic disease may have so altered the blood and the secretions of the various organs as to render them incapable of supplying food for the nourishment and development of germs.
>
> 4. The means of conveying poisons are often absent simply by virtue of the adoption of a system of perfect isolation; and although the germs may be carried by the atmosphere, the latter may be so charged with ozone, or be so attenuated (as in high elevations), as to destroy their vitality, or to fail in carrying them.
>
> Referring more particularly to pleuro-pneumonia zymotica itself, we find that many cattle, although placed under circumstances never so favorable for its reception, withstand the effects of the poison altogether; others suffer but very slightly from it; while in a byre it frequently develops itself first in the cow which is the farthest removed from the one which introduced the disease; not only this, cows in byres several yards distant from the one into which the disease has been introduced will frequently become the victims of the disease, and those cohabiting with the affected animal escape.

Again, a lot of cattle may be purchased, and subdivided into several smaller lots, and sent to different parts of a district or farm; in due time the disease may break out in one or more lots and the remainder never show a symptom of it.

A relation of one well-observed outbreak will sufficiently illustrate the remarkable vagaries sometimes exhibited in the spread of the disease:

P. W. had six cows in his possession for six months; no fresh animals were introduced during that time, nevertheless the disease made its appearance at the end of the six months. Whence came it? Necessarily, by spontaneous generation, or from one of the following sources, viz., through the medium of fomites, from the retention of virus in the cow shed, or from the retention of virus in a latent form in the system of one of the animals.

A case of zymotic pleuro-pneumonia occurred in his byre on the 3d of April, 1876, about which date other premises were rented, a new shippon erected, and immediately adjoining a double-stalled shippon or turnip house. The outer partition and upper part of the front wall of the latter consisted of boards taken from the old byre. Two cows (one of them twelve months in), the remnant of the old stock, were removed to the new shippon, and, on the 25th April, two others were purchased at Hawick, one of which was placed in the byre above referred to as being composed partly of boards from the old one, and on the 14th August (four months after purchase) was attacked by zymotic pleuro-pneumonia; the other was stalled between the old cows, and did not contract the disease. I saw one of the two old cows above referred to (a Dutch cow) eight or nine months after the date mentioned; she was perfectly healthy, and although stalled in the midst of the disease, proved refractory to the virus.

A very important question in connection with the spread of this disease is the possibility or otherwise of an animal exhaling the virus during convalescence, or for any period after apparent recovery. I am, myself, convinced that the disease is propagated not only during convalescence but for a tolerable length of time afterwards, especially as the diseased portion of lung communicates with a bronchial tube, and several instances have come under my observation in which animals said to have recovered from the disease were the means of introducing the infection into healthy byres. I have also known instances in which old stagers have been kept among stocks of cows for considerable periods, the disease all the time continuing its ravages, and after the slaughter of the affected animal or animals the malady has ceased.

In a paper which I read on the subject several years ago at one of the meetings of the Scottish Metropolitan Veterinary Medical Association, I brought the question of propagation by convalescents prominently forward, and one of the main arguments I then used in favor of the view expressed was the fact that in the case of several convalescents which I had had the opportunity of watching I had found a permanent temperature of 103° F., and that these animals were very susceptible to the action of adverse influences. Since that time I have seen several instances in which animals having recovered from the disease, although never re-exposed to the contagion in any way, have developed secondary zymotic pleuro-pneumonia, either in the healthy lung or the healthy portion of the diseased lung, after the lapse of a considerable period from the primary attack.

I find, too, other evidence from two independent sources which strongly favors the view that the disease is propagated by convalescents.

This evidence is to be found, *a*, in an article in the *Veterinarian*, by Professor Ferrein, of Berlin; *b*, in the Annual Report of the Veterinary Department of the Privy Council Office for 1876.

A case illustrating the above statements in a remarkable manner is given by Dr. Zündel, veterinarian of Elsass-Lothringen, in his report for 1881-'82, p. 37. Dr. Zündel is one of the best known and ablest veterinarians of Europe, and his writings are everywhere received with the greatest confidence. Pleuro-pneumonia was announced in October, 1881, in Oberdorf, and on investigation it was found to have been introduced by a cow from Grenzingen. In this case it was 3½ months from the time the animal was exposed until she contracted the disease, and three other animals were infected by her. Before it was ascertained that this cow was affected with pleuro-pneumonia she was allowed to pasture in the same field with seventeen other cattle from six different stables; all of these were kept under observation, but not one contracted the disease.

It would be easy to multiply examples of this kind from European literature and to compile a volume of them if necessary, but it is simply my purpose to show that the pleuro-pneumonia of Europe is identical in all its characters with the pleuro-pneumonia of America, and for this purpose no further quotations are needed.

The losses from the disease are never immediate and widespread on its first introduction into a country; from the nature of the case this is impossible, because from two to three months pass between the first exposure and the sickening of the infected animals, and a second period of equal length must elapse before a second lot of animals can become diseased from the first. During this time the cattle plague or foot-and-mouth disease would have run through the herds of a large section of country, produced what damage they could, and disappeared, and the stockman would then be free to carry on his business until there was a fresh importation of contagion. Not so with pleuro-pneumonia, however, for this disease, when once introduced into a herd, remains for months and years, not causing such alarming losses at any one time, but in the aggregate doing more damage than the terrible cattle plague.

The long preservation of the virus in buildings and pastures, and in the lungs of animals which have had a mild attack and apparently recovered, gives to pleuro-pneumonia a peculiar danger and makes it more difficult to eradicate than any other disease. These very facts show the great importance of preventing the spread of the disease while by far the greater part of our territory is still free from the infection. Once carried to the dairies surrounding the Western cities, or introduced into the Western stock-yards, and, above all, once scattered over the great ranges of the Territories, it would be continually carried into the channels of commerce, and our losses would not only be enormous, but it would be found a work of many years with the most vigorous policy to exterminate the plague. Pleuro-pneumonia has remained in small sections of other countries, notably in France and Swabia, for as long a time and attracting as little notice as it has in America, but the time has finally come in all of these when, by a change in the condition of the cattle trade and other favorable conditions, the disease was carried far beyond its original haunts to many new centers almost simultaneously, and almost before this extension could be realized it had gone too far to be remedied. The recent outbreak in the Western States is an example of how easily the same thing may occur in this country. The disease had existed for eight months west of the Alleghanies before it was discovered; it had been carried to three different States, and exposed cattle had gone to seven different States, but fortunately in the majority of cases they did not carry the disease with them. Within a short time a combination sale would have been made in Chicago into which were entered some twenty animals from two thoroughly infected herds, and from here the disease would doubtless have been scattered to every part of the West. It was only by a number of the most fortunate coincidents that the contagion was not more widely spread than has really occurred. A considerable number of animals were purchased from the infected herd at Virginia by parties in Nebraska, but these were resold to parties in Illinois and Missouri; eighteen were taken to Western Missouri, but these did not scatter the contagion; other animals from infected herds went to Iowa, Mississippi, and Texas, and these again remained free from disease. Such cases, where contagion is not carried by animals from infected herds, are common with the disease in Europe, as has been shown above, but it is evident that the chances were just as great of its being carried by the cattle which went to Texas or Western Missouri as by those which went to farms in Illinois and Kentucky. We cannot avoid the conclusion, therefore, that there was a narrow escape from the irremediable infection of the West, and it would seem that this vivid illustration of the danger by which the great cattle industry is menaced should be sufficient to secure better protection for the future.

THE CONTAGIOUSNESS AND MORTALITY OF PLEURO-PNEUMONIA.

The results obtained by the able commission appointed by the French Government in 1850 are among the most trustworthy that have ever been recorded. On this commission were some of the most able scientific men of the century—such men as Bouley, Magendie, Rayer, Cl. Bernard, Renault, and Baudement; and their report is a model in clearness of statement and in the value of its conclusions. In all, 46 animals were exposed to the contagion; 15 of these plainly contracted the disease, of which 4 died and 11 recovered; 10 had a slight indisposition of short duration, but without positive symptoms of pleuro-pneumonia; 21 showed no symptoms of disease during life, but when slaughtered and examined it was found that 6 of them had had an attack of the disease. As the disease is usually diagnosed even by veterinarians it would be said that of the 46 animals exposed 15 contracted the disease and 31 were insusceptible. As a matter of scientific fact, however, 31 contracted the disease and but 15 were insusceptible. This result illustrates the great source of danger from pleuro-pneumonia, for while in round numbers two-thirds of the animals exposed contracted the malady, one-third of those exposed were affected in so mild a form as to escape detection, but they still had the disease in their lungs and were capable of transmitting it to other animals.

For the purpose of comparison with American outbreaks, we must leave out of consideration the cases that were too mild to be noticed by the veterinarian before the slaughter of the animals, and we would therefore have the following result from these experiments:

	Number.	Per cent.
Animals exposed	46	....
Contracted pleuro-pneumonia	15	32.61
Were insusceptible	31	67.39
Died	4	8.70
Recovered	11	23.91

The following table is compiled from the official statistics of the various European countries mentioned, and contains all at hand in which complete data are given:

Country.	Year.	Exposed.	Sick.	Died.	Slaughtered.
Elsass-Lothringen	1881-'82	29	16	1	28
Bavaria	1881-'82	3,315	660	21	987
Prussia	1881-'82	10,154	1,856	39	1,943
Prussia	1882-'83	8,875	1,953	48	2,031
Prussia	1883-'84	11,171	2,750	81	2,989
Kingdom of Saxony	1882	471	55	2	53
Kingdom of Saxony	1883	667	159	4	193
		34,682	7,449	196	8,224

It will be seen from this table, which embraces the careful observations made upon more than 34,000 head of cattle which had undoubtedly been exposed to the genuine European lung plague that the proportion which sickened there was 21½ per cent.; and with so large a number of observations this proportion cannot vary greatly from the general average of Europe. There may be, however, a very considerable variation from this percentage in certain years or in particular localities, for we see that in the Kingdom of Saxony in 1882 but 12 per cent. of those exposed contracted the disease; while in the following year in the same kingdom this proportion was 23.8 per cent., or about double.

In the United States we have only a small number of cases recorded which we can use for comparison with the above. In the report of the secretary of the Massachusetts Board of Agriculture for 1860 (p. 70) it is stated that the number of animals which had been exposed and the number which died before the commissioners commenced slaughtering was as follows, no mention being made in regard to the number which had been sick and survived:

Herd.	Exposed.	Died.
Chenery's	67	27
Leonard Stoddard's	48	13
Alden Olmstead's	23	7
C. P. Huntington's	22	8
A. A. Needham's	22	8
A. B. Woodis'	21	4
Total	203	57

In New York, in 1878 and 1879, the following figures were given by Professor Law in regard to a few herds:

Herd.	Exposed.	Affected.
Blissville stables	600	164
Mrs. Kelly, Long Island	3	3
Mrs. Robertson, Westchester County	12	5
Mr. Carr, New York City	5	3
Mrs. Erath, New York City	9	5
P. McCabe, New York City	9	9
Total	638	189

Using the above figures with those already given in regard to Pennsylvania and Connecticut, and the cases which have occurred in the recent outbreak in the West where, according to latest information, 105 animals have been affected, and we have the following table of American cases:

Outbreaks.	Year.	Exposed.	Affected.	Percentage.
Massachusetts	1859–1860	203	57	28.1
New York	1878–1879	638	189	29.6
Connecticut	1883	9	7	77.7
Pennsylvania	1883–1884	296	143	48.3
In the West	1884	627	105	16.7
Total and average	....	1,773	501	28.2

That is, while the proportion of animals exposed which contracted the disease in Europe averages 21.5 per cent., the average in the United States, so far as we are able to determine from the number of cases recorded, is 28.2 per cent. This difference is so slight that we may conclude that the disease here is identical in its character with that of Europe, and so far from losing any of its virulence, like most other contagious diseases, it has rather increased its destructiveness by invading fresh territory.

CHARACTERS WHICH DISTINGUISH CONTAGIOUS PLEURO-PNEUMONIA FROM OTHER DISEASES.

The characters of contagious pleuro-pneumonia, or lung plague, are so distinct from other diseases that it is not difficult for a veterinarian of experience to make a correct diagnosis after a thorough investigation has been made. Tuberculosis and some other chronic affections of the lungs are in some cases difficult to distinguish from chronic pleuro-pneumonia, but when acute cases of the latter are found, and particularly when animals are slaughtered for examination, there is no longer a possibility of a mistake. Ordinary acute inflammations of the lungs occur in winter and spring; but a few animals in a herd are affected; these cases all occur at about the same time, and there is no connection by movement of animals with other diseased herds. Lung plague, on the other hand, occurs at all seasons of the year, in summer as well as in winter, and is rather more fatal during the hot weather of summer than at any other time. With this disease, the different members of the herd are successively attacked, with intervals of a few weeks, or even months, until a large proportion has been affected, and the history of the introduction of the disease with an animal from another infected herd can generally be traced.

The following conclusions were reached after a full discussion by the International Veterinary Congress, held in Brussels, in 1883, in regard to the diagnosis of this disease. In this congress were present the leading veterinarians of the world, and these conclusions may therefore be received as representing the best views which the knowledge and experience of the present age can suggest. Nearly all of the principal official veterinarians of Europe who are charged with the duty of combating contagious diseases were present and assisted in formulating the expressions of the body.

1. From the anatomical standpoint, at least as regards veterinary police, should be considered as epizootic contagious pleuro-pneumonia, every lobular pleuro-pneumonia which is at the same time interlobular, and the development of which does not depend upon local traumatic causes.

2. Considered from the physiological standpoint with the living animal, epizootic contagious pleuro-pneumonia is characterized particularly by contagion and the symptoms of lobular pneumonia.

3. Should be considered: *a.*—As suspected of epizootic contagious pleuro-pneumonia every animal which in an infected locality presents either a fever of reaction or symptoms of a disease of the chest.

b.—As suspected of contamination, every animal which is found in an infected stable, or which has been in one within three months, or which may have been contaminated in any other manner.

These are in brief the characters which we have relied upon in diagnosing the disease in this country, and the reasons which have been regarded as sufficient to justify suspicion in cases that were not well marked. At the present time, when the profession in the different parts of the world is so closely connected by journals, veterinary works, and by postal facilities, there is but little difference in the opinions of its most advanced members wherever they are located. The literature of the world is now at the command of educated veterinarians in all countries, and as a consequence the knowledge of every section soon finds its way into the universal fund; facts and conclusions brought out in discussions are read the world over, and the views which are most clearly in accordance with the facts are soon accepted everywhere. A summary of the more important points of difference between pleuro-pneumonia and ordinary inflammations of the respiratory organs is given below:

Contagious pleuro-pneumonia.	*Sporadic inflammation of lungs.*
As frequent and fatal in summer as in winter, and occurs with the best surroundings and care.	Seldom or never occurs in summer; properly arranged stables and good care prevent it at all times.
Not greatly influenced by the weather or climate; most fatal in summer and in warm climates.	Most frequent and fatal in cold climates, and in winter and early spring when weather is cold and changeable.
From one-third to two-thirds of whole number of animals in a herd usually affected.	Seldom more than one or two affected in a large herd.
Intervals of one week to three months between the development of different cases.	The attacks are simultaneous.
History of contagion can generally be traced.	No history of contagion.
Confined to an infected district, and cases do not occur outside of such district except where contagion is carried by movement of cattle.	Confined to no particular district or section of the country.
Inoculation confers immunity.	Cannot be prevented by inoculation.
Insidious in its development; often slow in its progress.	Sudden in development and rapid in progress.
Highest temperature generally reached several days before there is appreciable hepatization.	Highest temperature not reached until hepatization has reached greatest extent.
Is but slightly influenced by medical treatment.	Yields readily to medical treatment.
As a rule, no resolution in the inflamed lung.	Resolution usually occurs.
Exists three or four weeks before apparent recovery.	Runs its course in from five to twelve days.
When fatal, death generally occurs after a sickness of from two to four weeks.	Death occurs within eight days as a rule, in fatal cases.
From thirty to fifty per cent. of cases terminate in death.	Very few cases fatal.
A whole lung frequently inflamed, and often a considerable part of both lungs.	Confined to a limited portion of one lung.
Inflammation progressive; autopsy clearly shows that different lobules have been successively invaded.	Inflamation generally limited to part originally affected.
Distension of interlobular connective tissue with exudation material, and difference in coloration of lobules produces very plain marbleized appearance.	Marbleized appearance absent or much less distinct.
False membranes and pleural adhesions seldom, if ever, absent.	False membranes and pleural adhesions not common.
Effusion of liquid into the pleural cavity in nearly all cases.	Effusion rare.

THE DANGER FROM PLEURO-PNEUMONIA.

That pleuro-pneumonia does not spread beyond an infected district, except by the movement of cattle, has been shown by our experience during the past twenty years to be as true in this country as I have demonstrated in the preceding parts of this report that it has been true in Europe. With the increase of traffic, however, the danger of spreading the disease increases, and with such profound modifications in the animal commerce of a country as occurred on the continent of Europe from 1790 to 1815, and in Great Britain between 1840 and 1850, it is difficult or impossible to prevent the spread of this disease when it exists in any part of a country.

In the United States, so long as there was little, if any, movement of cattle from the infected districts of the East toward the uninfected States of the West, the plague was not carried across the Alleghany Mountains. The earlier shipments consisted mostly of thoroughbred animals, and these went from herds, the owners of which had a reputation to sustain, and consequently in the rare instances, when such herds became infected, no shipments were made. More recently, large numbers of eastern calves have been going to the West, gathered up at first from Ohio, Western New York, and Western Pennsylvania, but as the demand becomes larger and the price increases they are drawn from farther and farther East, until now some of them are obtained dangerously near to infected districts.

A trade far more dangerous, however, has been built up largely within the last two or three years, and consists in buying grade Jersey cattle wherever they can be found the cheapest, and carrying them to the West, where they are scattered among valuable herds, and are even sent by the car-load to the plains of Texas. The prices have ruled so high for such animals in the various Western States, and there have been such large profits in the business that the whole eastern section of the United States has been hunted over for grade Jerseys. At first they were taken from the New England States, but as the demand increased and the supply in these sections was exhausted, dealers turned to the infected sections, where they could buy even cheaper, hoping, by their shrewdness, to escape loss. With any other disease this might be possible, but with the insidious and deceptive lung plague it was only a question of time when it would be carried to the West.

About a year ago a number of grade Jerseys were gathered together in the vicinity of Baltimore. There is now no means of learning from what stables these were purchased, but almost beyond a doubt they carried the disease to Troy, Ohio, from which point it was distributed directly and indirectly to the various herds which were found during the past summer to be infected in the West. Other Jersey cattle were purchased by Mr. Dye at about the same time, but they were registered animals, and we know from what herds they came, and that these were not infected with pleuro-pneumonia. On the other hand it is certain that many of the herds in the locality where the grades were obtained are infected with pleuro-pneumonia, and that the losses from this disease there are continuous and heavy.

The stockmen of the West have good reason for congratulating themselves that they have escaped so well from this outbreak. Cattle from infected herds had been sent to Mississippi, Texas, Missouri, and Iowa, without carrying the contagion; others had been purchased for Nebraska, Tennessee, and North Carolina, but had not been shipped; a large combination sale, in which twenty or more animals from infected herds had been entered, would have been made at Chicago about September 1, if the discovery of this disease had not previously occurred. In all of these respects the country was fortunate, but it is a mistake to suppose that the disease did not make rapid progress during the time it existed in the West. Over one hundred cases produced by contagion from one animal within eight months is as much progress as we should expect from pleuro-pneumonia in such a period. If the progress had continued at this rate, and each one of these one hundred animals had infected one hundred others during the next eight months, we should have had by the end of that time 10,000 animals affected with the disease; in another period of eight months at the same rate the number would have reached 1,000,000, and it is easily seen that we have not half enough cattle in the United States to furnish the victims that would be required to carry this rate of progress through a fourth period of the same length.

Fortunately, pleuro-pneumonia seldom continues spreading so rapidly for a very long period unless the conditions are peculiarly favorable for its dissemination. If it had been carried to the plains of Texas with some of the grade Jerseys, which have been shipped there and turned out upon the ranges, it would doubtless have been scattered by the herds driven from there through the various Territories of the West and Northwest, and in this way have surprised the country by the magnitude of the losses which would have been caused. Once planted on the ranges it would have been continually carried eastward by beef cattle and by stock cattle; it would infect the stock-yards of our great cities, and there would no longer be safety for the bovine race in any part of the country. This, it seems to me, is the great danger which should be guarded against while it is yet time. True, to the best of our knowledge, this disease has never been carried to the ranges of the Western States and Territories, but as I write this I remember that less than a year ago I wrote in a report that pleuro-pneumonia had never been carried west of the Alleghanies, and yet at that very time it had been carried there and was smouldering, ready to break out and extend itself, as it did, within a few months, to three States and a dozen different herds. In the present condition of the market this disease is nearly as likely to be carried to Texas as to Ohio or Illinois, and consequently this recent outbreak demonstrates that there is danger of its being carried to the unfenced ranges of the West where the restrictive measures which have stopped its progress in Ohio, Kentucky, and Illinois could not be enforced without an enormous expenditure, if at all.

It is useless to expect that pleuro-pneumonia will be robbed of its destructiveness by the pure and dry climate of the high altitudes in which the greater part of the western ranges are situated. Those acquainted with the history of this disease cannot fail to be incredulous in regard to this result, for there is nothing to favor such a view to be found in the records of any time or any country. Why, the early home of pleuro-pneumonia in Europe was the mountain ranges of the interior of the Continent, and particularly of the Alps, where it has remained for centuries and exists to-day in spite of the measures adopted for its suppression. Again, it has been carried over the plains of South Africa to the interior of that Continent, and instead of losing its destructive characters it appears to have acquired greater virulence.

The inhabitants of our western plains, heretofore preserved from the great losses incident to contagious diseases because these have never yet been introduced there, evidently attribute too much of their good fortune to the sanitary influence of their incontestably healthful climate. A year ago they told me with equal assurance that Texas fever could under no circumstances affect the cattle on the dry and elevated ranges of Colorado and Wyoming, and yet the past summer has produced the most ample evidence of their mistake and demonstrated that this disease is just as fatal there as it is in other parts of the country. The same will undoubtedly prove true with pleuro-pneumonia when the opportunity for the demonstration arrives, but unlike Texas fever it will not run its course in a few weeks and disappear, nor will its germs be destroyed by the frosts of winter.

MEASURES ADOPTED IN EUROPE FOR SUPPRESSING CONTAGIOUS PLEURO-PNEUMONIA.

During the half century or more which followed the introduction of the lung plague into the better parts of the various countries of Europe, almost every possible plan has been adopted for its control rather than slaughter all infected animals. Various methods of medical treatment, inoculation and quarantine have been tried and found insufficient to protect the cattle from this disease. At present the tendency is to slaughter all animals which are sick or which have been exposed to the contagion, and thus stop at once the multiplication of the virus from which alone new cases of disease are produced. The treatment and cure of sick animals has been found much more expensive than their slaughter, because even after recovery they are dangerous and liable to cause new outbreaks of disease wherever they go. Inoculation, while it has protected a certain proportion of the inoculated animals, has not greatly lessened the prevalence of the disease. The following is a summary of the measures now in force in the more important countries:

Great Britain.—All diseased animals are slaughtered, and exposed ones may be if local authorities think fit.

Prussia.—All diseased animals are slaughtered, and suspected ones may be ordered destroyed, according to the judgment of the higher officials.

Austria.—Diseased animals separated and quarantined.

Holland.—With exception of the "Spœlingsdistrikte" the sick animals and most of the suspected ones are slaughtered. Within the "Spœlingsdistrikte" slaughter is only enforced to a limited extent; inoculation is compulsory.

Belgium.—All sick animals destroyed.

Switzerland.—All sick and exposed animals are killed. By special permission, in certain cases sick or exposed animals may be preserved under strict quarantine regulations, but such animals must be finally slaughtered and are, under no circumstances, allowed to enter into the commerce of the country.

Kingdom of Saxony.—All sick and suspected animals are slaughtered.

EXTENT OF PLEURO-PNEUMONIA AND THE IMPORTANCE OF NATIONAL ACTION IN REGARD TO THE CONTAGIOUS DISEASES OF ANIMALS.

The following statement was prepared for the use of the House Committee on Agriculture in January, 1884:

The extent of territory infected with contagious pleuro-pneumonia of cattle and the number of animals actually suffering from this disease, are insignificant in comparison with the annual direct and indirect losses traceable to it, and the danger to which our immense live-stock industry is continually subjected.

In Connecticut two herds were infected during the past summer, in which 12 animals were exposed and 7 contracted the disease. In one of these herds the affected animal was destroyed, and at last accounts no others had contracted the disease; in the other herd 4 animals had died, or had been killed, and 2 with very extensively diseased lungs remained in quarantine. Both of these were Jerseys, and the owner refused to have them destroyed. What has been done with them, or what will be, I am unable to say, as the State authorities seem powerless to proceed beyond quarantine, and this seems to have been by no means secure.

In the State of New York, although the disease is almost entirely confined to the western end of Long Island, to Staten Island, and New York City, these localities are quite extensively infected, and as there are more than two thousand stables, some of which contain several hundred cows, and many of which contain from 50 to 100, it is the most dangerous district in the country at this time. Recent reports are to the effect that the disease is extending through the river counties, and exists in herds located from 50 to 60 miles north of New York City. How many cattle are affected in these counties I am unable to say, but the existence of the disease here is really of much greater importance to the country at large than the number of diseased animals would lead one to suppose, because it is a district where many thoroughbred cattle are raised and from which they are shipped to all parts of the United States.

New Jersey was recently supposed to be nearly free from pleuro-pneumonia, but the fact that a number of cases occurred without the knowledge of the State authorities, that a still larger number of herds were lately known to be infected in Union and Essex Counties, and that a very extensive outbreak in Hunterdon County was recently traced by means of sick cattle shipped to the New York market, and discovered by the inspector employed by the United States Department of Agriculture who is stationed at Jersey City, leads to the suspicion that a thorough inspection of the State might bring to light still other cases. The Hunterdon County outbreak was one of the most extensive that has recently occurred. It was supposed to have originated from a car-load of cows brought from Pennsylvania; but where these were infected is not known. Seven herds, at last accounts, were in quarantine; and as all were large herds, containing from 40 to 70 cattle, a large number of animals were exposed.

Inoculation was extensively practiced to check the fatality; but, in spite of this, reliable authority places the loss at over 50 head.

In Pennsylvania there has recently been another very extensive outbreak, which was the result of taking a car-load of 14 cows from the Calvert stock-yards in Baltimore to Chester County. Most of these cows were taken into large dairy herds, which they thoroughly infected. In each of these cases the Baltimore cows were the first to sicken, and a large proportion of the native cattle were soon affected with the same disease. These herds were visited the 3d of October by the Veterinarian of the Department of Agriculture, in company with the State authorities, who killed 8 of the animals in his presence in order to satisfy him as to the nature of the disease. The cases were typical cases of pleuro-pneumonia, and all those appearances were present which were recently accepted by the International Veterinary Congress held at Brussels as characteristic of contagious pleuro-pneumonia. In most cases a whole lung was hepatized; the inflammation was of different ages, showing the progressive character of the disease; the interlobular tissue was greatly distended with the exudation, and the pleurisy was intense. According to an official report, dated October 30, the number of animals known to have been exposed was 104, and the number of sick ones that had been killed or had died was 46. A semi-official report of the present month places the number destroyed at 70. It is now believed that the disease has been entirely overcome, and that the State of Pennsylvania is free from it.

In Maryland and the District of Columbia there are many infected herds in which a comparatively large number of animals annually contract the disease. By direction of the Commissioner of Agriculture a reliable Inspector was sent to Baltimore late in October to learn the condition of the stables there as regards this disease.

Nineteen stables, containing 398 animals, were examined. In twelve of these the infection was admitted; one had lost more than 200 animals within three years; others had lost heavily for years; 12 sick animals were found, 18 recent deaths were admitted, and 3 sick cows had just been sold or exchanged. This number of stables comprises but a small part of those in the vicinity of Baltimore, but it is believed that the number is sufficient to demonstrate the presence and dangerous character of the disease. We have no information of pleuro-pneumonia in the country districts of Maryland at any great distance from the cities.

One or more herds near the District of Columbia have recently lost a number of cows, and at latest accounts had some sick. Within the District, without making any regular inspection, three infected herds have been found where from 3 to 6 animals are admitted to have been lost within the year. In Virginia there are stables from which animals have recently been lost with symptoms of this disease; but none of these could be secured for examination, and therefore we can not be positive in regard to the nature of the disease.

REASONS FOR BELIEVING IT CONTAGIOUS.

The first great reason for believing this to be contagious pleuro-pneumonia is the fact that nowhere in the country outside of the comparatively small strip of territory stretching from Connecticut to Virginia, and east of the Alleghany Mountains, have any cases been found which bear any close resemblance to the disease under consideration. If this disease were the result of climatic causes, or if it were produced by improper food and care, then we should certainly find it distributed over the whole country, or at least in all of those parts of it where similar conditions exist. It cannot be originated by the manner of stabling and feeding cows near our Eastern cities, for substantially the same conditions exist at Rochester, Buffalo, Cleveland, Detroit, Chicago, Saint Louis, Cincinnati, and other Western cities, and no veterinarian has been able to find any similar cases of disease there, although special inspection has been made by competent persons.

The disease is not confined to stable cows, however, nor to those seasons of the year when acute lung diseases can be accounted for by the inclemency of the weather. The outbreak referred to in Connecticut occurred in the summer, in a country district, and where the cattle were running upon nice pasture fields. The extensive outbreaks in New Jersey and Pennsylvania also happened in summer, and were in the best farming districts of these States.

In this connection attention is called to the fact that in the State of Pennsylvania about ninety herds have been infected since March, 1879, and that notwithstanding the appointment of special agents in every part of the State, and the investigation of all cattle diseases wherever found, there was no disease resembling pleuro-pneumonia discovered except in eight of the sixty-seven counties of that State. The remaining fifty-nine counties have been free from any suspicion of this plague. What is even more significant is the fact that these counties are not distributed over various parts of the State, but that they join each other, and are all in the southeastern corner of the State, where there is the greatest danger of infection by cattle brought from Philadelphia and Baltimore. With seventeen of these herds the infection was traced to cattle from Baltimore or other points in Maryland; with twenty-one it was traced to Philadelphia; with ten it was traced to cattle from herds in Pennsylvania known to be diseased.

The most favorable conditions of life were not sufficient to protect the cattle where this disease was introduced. I have already mentioned that a number of the outbreaks referred to occurred during the summer, and that the animals were running upon irreproachable pasture fields. Many of the affected cows were young and in fine condition. In Connecticut a Jersey bull, less than two years old, and two steers fit for beef, were among the victims. Again, the disease as we see it here does not occur in isolated herds a single case at a time, as does non-infectious lung disease, but when it enters a herd a majority of the cattle are affected sooner or later. Some of the herds in Brooklyn and Baltimore have been losing cows from this plague for years, and one near the latter city, where but about fifty cows were kept at a time, has lost between 200 and 300 cows within three years.

These instances, all recent, are referred to, not as all the evidence bearing on this point, but simply as examples of what has been occurring for years past; and it is believed that they cannot be explained on any other hypothesis than the contagiousness of the disease.

DANGER GREATER THAN EXTENT OF INFECTED TERRITORY AND NUMBER OF DISEASED ANIMALS WOULD INDICATE.

Glancing over the territory which I have stated to be infected, it must be confessed that it is not extensive—a single farm with perhaps five animals in Connecticut, about four counties in New York, as many in New Jersey, two or three counties in Maryland, and possibly a few stables in Delaware and Virginia.

In most of the infected herds there are but one or two sick animals at a time, and frequently there are none; for where the disease has existed for a certain time the susceptible animals die off and only those which possess a certain immunity from it remain.

As about 20 per cent. of all the animals exposed are able to resist the contagion indefinitely, a herd of comparatively insusceptible cattle is in time acquired, and the time necessary for this is shortened both in Baltimore and Brooklyn by the practice of inoculation.

But these stables and grounds remain infected, and a large portion of the new cows brought into them contract the disease unless they are previously protected by inoculation. The practice of inoculation does not destroy the infection; on the other hand it keeps it up, but it enables dairymen to keep their cows in infected stables without great loss, when without it more than half of the new cows brought into them would surely die.

Another fact of great importance brought out by the experiments of the French pleuro-pneumonia commission is that about 30 per cent. of the animals exposed to this disease show no symptoms of it beyond a slight cough. Such animals are probably as dangerous to others as those which have it in a more severe form, and yet they can be transported to various parts of the country without exciting the least suspicion.

The animal which is supposed to have caused the outbreak in Connecticut was probably in this condition, as a careful examination of her lungs did not enable the veterinarians to detect any evidences of the disease; and yet pleuro-pneumonia existed in the stable from which she came, and her admission into the new herd was followed by the seven cases that have been mentioned. Similar instances are referred to again and again by the veterinarians of every country where the disease exists.

These infected districts, though small, are then a real danger to the whole country, because all the way from Connecticut to Virginia there is a large and increasing number of herds of thoroughbred cattle, which are frequently shipped to the West and some of which have from time to time been infected with this disease. Fortunately, the owners of thoroughbred cattle have generally had too much regard for their reputation to ship cattle when there was any disease in their herds, and the common cattle have not been sent to a sufficient distance to do much harm.

But with the increased price of cattle a large number are being shipped from the East toward the West, and the danger of carrying the disease is consequently increasing. If the car-load of cattle shipped from Baltimore to Chester County, Pennsylvania, had gone to the ranges of the West, they might have done irreparable harm. Again, the thoroughbred Jersey cow which went from an infected stable in New Jersey might as readily have been shipped to the West; and I have been informed that if the Connecticut outbreak had occurred a few months later one or more of the herds would have been sent, according to contract, to a Western State. Now, while it is true that pleuro-pneumonia has existed in the East for forty years without having been carried to the West, it must be admitted, from what has occurred so many times in Pennsylvania and Connecticut, that there has been danger of this, and that this danger is increasing with the larger number of cattle now being shipped in that direction. No doubt this danger has been exaggerated, but the fact that there is danger, and that the disease once carried to the Western herding grounds would probably be beyond our control, if we can judge from the experience of Australia and South Africa, is sufficient to show the importance of grappling with it while it can be so easily handled. The rapidity with which a disease spreads on these ranges, when once introduced, is illustrated by an occurrence of last summer in Southwestern Texas. A drove of cattle brought a communicable disease to that section, which the army surgeons believed to be contagious pleuro-pneumonia; but before any careful examination could be made several hundred cattle had died, and a large territory was infected. Fortunately, investigation showed that this was not pleuro-pneumonia, but a disease which does not outlast a single season of the year. If it had proved to be pleuro-pneumonia, would it not have been a national calamity? With a large territory already infected, with no money and no power to control the disease, and occurring in summer months, before the State and national legislative bodies would convene, it is difficult to see how any effective measure could have been adopted.

THE INEFFICIENCY OF STATE ACTION.

Though a number of attempts have been made by the States now infected to rid themselves of pleuro-pneumonia these have generally or always failed, because for various reasons the work was not thoroughly done. We saw the State authorities of Connecticut unable to exterminate the disease a few months ago, when but a single herd contained sick animals. The stables of Brooklyn were never under complete supervision, and some could not be entered by the inspectors even when the State of New York was most active in its endeavors at extirpation; and though the authorities of New Jersey have been engaged at the same task for five years, the State has probably never during that time been entirely free from pleuro-pneumonia. In Maryland the assertion has been made again and again that there were no cases of this disease in the State, and yet during any part of this time a thorough inspection could not have failed to reveal a considerable number. At best the attempts of the States have been spasmodic; and while one State was earnestly striving to accomplish something a neighboring one would allow the shipment of diseased cattle, and counteract the influence of the former. As a rule, therefore, State action has never been thorough, and the lack of unity of action between the States has prevented any lasting benefit even when much has been accomplished.

ADVANTAGES OF THE WORK BEING DIRECTED BY THE UNITED STATES GOVERNMENT.

A national direction of the work for the extermination of pleuro-pneumonia would overcome at once the discouraging features which have done so much to prevent the efforts of the individual States from being effective. With inspections in every infected State the shipment of diseased cattle would soon cease; new outbreaks would thus be prevented, and the danger which has so long menaced the great cattle interests of the country would be removed. The work would be more thorough and energetic, because those engaged in it would not be directly or indirectly dependent upon the good-will of the interested cattle owners for their positions, and the plea of inability to pay for the diseased cattle which ought to be slaughtered would also be overcome. These have been the principal obstacles to the success of State action, and practically they are so great as to make it next to impossible for the States alone to free themselves from this plague.

THE PRESENCE OF PLEURO-PNEUMONIA COSTS ANNUALLY MORE THAN WOULD BE NECESSARY FOR ITS DESTRUCTION.

Owing to the presence of pleuro-pneumonia in the United States, every steer shipped to Great Britain must be slaughtered within a certain time on the wharf where he is landed. This restriction upon the export cattle trade is said by competent authorities to make the price of our steers average $10 less than similar animals shipped from Canada. With over 100,000 beeves going abroad every year, this makes a loss of $1,000,000 annually, or enough to clear our country of the disease. Besides this, there are the continual losses which are going on in the infected districts, and the disturbed condition of trade from the many false alarms in regard to the spread of this disease, the entire annual losses being estimated by good authorities as high as $3,000,000.

IMPORTANCE OF INVESTIGATING OTHER DISEASES.

The proposition of establishing a permanent bureau for investigating the communicable diseases of animals is a matter of the greatest importance. While we have no more disease than other countries in proportion to the number of our animals, the enormous development of our live-stock industry has made the question of contagious diseases one of peculiar interest to us. The cause of these plagues, which has been an impenetrable mystery during all the past ages of the world, is being revealed by the science of to-day, and the infinitely small organisms which are able to produce such terrible havoc in our flocks and herds are at last being brought under subjection themselves, and their study has revealed much of the greatest value to us in our warfare against them. A country with so much at stake, with millions of dollars annually swept away by this class of maladies, cannot afford to be idle. Other nations which have much less capital invested in animals than we have, see the necessity for this work and are making provisions for it; and it is to the credit of our country that we were one of the first to enter this field, and that results have been accomplished which will bear comparison with the investigations of any other country. But while much has been done, while millions of dollars have already been saved to our farmers by the facts thus far discovered, we have only made a beginning in the great work that is before us. Some of the most important diseases affecting our animals are still mysteries to us, and though they are distributed over large territories and decimate the live stock, we are ignorant of their cause; we do not know how they are kept up from year to year; we have no means of combating them, and the idea of freeing ourselves from their ravages has scarcely dawned upon us. A striking example of the necessity of such work is seen in the recent investigations of Texas cattle fever. This disease has been advancing and infecting new territory for a century, and until the last year or two we knew nothing about it, and our best informed veterinarians and stockmen did not suppose that it was found in one-fifth of the territory which it has actually overrun. These were points which it was necessary to understand before either legislative bodies or individuals could adopt intelligent measures for preventing the annual losses which have been most discouraging to the cattle industry in large sections of the country. And with every disease there are equally important points still to be investigated.

The laboratory and experiment station which have been fitted up during the past summer under the direction of the Commissioner of Agriculture, for investigating contagious diseases, make it possible to attempt the solution of questions which were formerly beyond our reach. The laboratory contains the most improved apparatus for such investigations, much of which was constructed according to new designs, especially for this work, and it is safe to say that the facilities here are now equal to those possessed by investigators of similar diseases in any country, and in some respects they greatly surpass them.

In conclusion, I would say there is not a department of original research or of agricultural investigation in regard to which there is more pressing need for development than this, and none which promises to effect a greater saving. Our losses are now heavy, but they must increase as our animal population increases, as new diseases are introduced, and fresh areas are infected. But it is not alone a question of dollars; the investigation of animal contagia must throw new light on those human plagues which in our country alone sweep a quarter of a million of human lives out of existence each year. Some of these animal diseases are communicable to man, and have a greater influence over our health and lives than is generally supposed, and any means of controlling them cannot fail to have an important influence on human health as well.

INVESTIGATIONS OF CONTAGIOUS PLEURO-PNEUMONIA IN NEW YORK, NEW JERSEY, AND THE DISTRICT OF COLUMBIA.

The disease made most prominent in the law for the organization of the Bureau of Animal Industry is the contagious pleuro-pneumonia of cattle, and as at the time of its passage there was considerable doubt among members of Congress, State and local authorities as to the existence of this disease in the United States, the first work to be done was evidently to make those investigations into the existence and nature of the diseases of animals in this country which were specially authorized in this law. The Government and local authorities of the States where pleuro-pneumonia was believed to exist were requested to cooperate with this Bureau and to grant its Inspectors authority to enter upon the premises of the citizens of those States in order to make the necessary inspections.

In the State of New York the Inspectors of the Bureau were authorized to act as Inspectors of the State Board of Health, and they also received the cordial assistance and cooperation of the Boards of Health of New York City, Brooklyn, and the other local boards of the districts in the vicinity of Brooklyn. With this assistance the Inspectors have been able to make a very thorough and complete investigation of the most populous districts of the State of New York where pleuro-pneumonia was supposed to exist. Some of the more sparsely settled sections of Long Island, where this disease has been reported, have not been inspected, owing to a lack of time and the limited number of employés allowed by law.

The New Jersey authorities have also cooperated cheerfully in investigating the condition of cattle in that State, but owing to the magnitude of the work in New York but a comparatively small portion of New Jersey has been gone over.

The governor of Pennsylvania and his special agent employed for the extermination of pleuro-pneumonia have expressed their willingness to cooperate with the Bureau in making an investigation, but no work has yet been done in that State.

Correspondence with the governor of Maryland justifies the belief that we shall have his cheerful cooperation to the full extent of the State law, but the details have not yet been arranged.

The Commissioners of the District of Columbia were requested to give our Inspectors authority to make an inspection of the cattle in Washington and vicinity, but such authority has not yet been granted. The Board of Health has cooperated with the inspectors of the Bureau, however, and a very satisfactory inspection has been made.

It may be remarked here that while all of these States have signified a willingness to cooperate in this investigation, there has been some objection to such an inspection upon the ground that more cases of disease would be found than the States could dispose of with their limited appropriations, and consequently it would cause much embarrassment to the State authorities. It has also been argued with much feeling that the publication of the number of cases existing in these States would create unnecessary alarm and interfere greatly with the commerce of the country, and particularly of the infected sections, without accomplishing a beneficial result since sufficient provision had not been made for exterminating the disease.

This inspection, moreover, does not give a correct idea of the number of cases which occur in a specified time in the district examined. There can be no doubt that the number of cases reported actually existed at the time the inspection was made, but if a tour had been made a month later we should probably have found as many at this new inspection, a large part of which would have been new cases. Again, many dairymen in all the large cities where this disease exists, have become very expert in the management of their cattle in order to avoid loss. The cows are kept in good condition for the butcher, and as soon as the first symptoms of the disease are seen the animals are sent to the slaughter-house. The cases which were found by our Inspectors were consequently only those which for some reason had not been, or could not be, disposed of in this way.

Considering these facts, and that probably not more than one-half of the cattle in the infected district have been examined, we must necessarily conclude that many more cattle in this infected district are annually affected with pleuro-pneumonia than are enumerated by our Inspectors. The exact number, however, cannot be determined until measures are adopted by which the movement of cattle from infected stables can be effectually controlled.

There have been a certain number of stables, though comparatively few in number, to which our Inspectors could not gain admittance; it is probable that all of these can be reached either by the special laws relating to this disease, or by those for the protection of the public health, but it was not deemed best to consume time in contesting such cases while this preliminary investigation was being made. There has also been much difficulty in making the inspections, owing to the fact that many of the cattle were on pasture when our Inspectors called to see them, and in a large number of cases the owners absolutely refused to bring them up for inspection, and as is well known, an inspection in the field for pleuro-pneumonia is of very little service. Generally a second visit was made to these places at a time when the cows were in the stable for milking, and in this way nearly all have been examined; but more time has been consumed than would otherwise have been necessary. Below will be found the inspections in detail:

INVESTIGATION OF CONTAGIOUS PLEURO-PNEUMONIA.

INSPECTIONS IN NEW YORK.

Date.		Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.								
July	31	Michael Abrams	502 11th avenue, New York City	1		Good	Good	C. B. Michener.
	31	Kaufman & Strauss	608 W. 40th street, New York City	2		do	do	Do.
	31	Alex. Odenheimer	610 W. 40th street, New York City	2		do	do	Do.
	31	Jas. O'Shea	511 W. 40th street, New York City	2		do	do	Do.
	31	Kaufman & Strauss	40th street, below 11th avenue, New York City			do	do	Do.
	31	Stern & Metzgar	do			do	do	Do.
Aug.	1	Jacob Shark	621 W. 23d street, New York City	2		do	do	Do.
	1	Michael Devine	521 W. 23d street, New York City	1		do	do	Do.
	1	L. B. Harrington	547 W. 24th street, New York City	1		do	do	Do.
	1	Wm. Fields	537 W. 28th street, New York City	4		do	do	Do.
	1	John McCallum	S. s. 30th street, between 10th and 11th avenues, New York City.	2		do	do	Do.
	1	David Stephenson	517 W. 40th street, New York City	2		do	do	Do.
	1	Patrick McIntyre	602 W. 40th street, New York City			do	do	Do.
	2	Mrs. English	627 W. 37th street, New York City	1		do	do	Do.
	2	Ellen Rafferty	602 W. 38th street, New York City	3		do	do	Do.
	2	Ambrose Sigswart	538 W. 40th street, New York City	7		do	do	Do.
	2	Mr. King	530 W. 43d street, New York City	1		do	do	Do.
	2	J. Rotzor	352 W. 43d street, New York City	2		Fair	Fair	Do.
	2	G. S. Asher	357 W. 45th street, New York City	1		Good	Good	Do.
	2	Jas. Miller	629 W. 46th street, New York City	1		Fair	Thin	Do.
	2	Wm. Kelly	609 W. 47th street, New York City	1		Good	Good	Do.
	2	John Higgins	616 W. 47th street, New York City	1		do	do	Do.
	4	Jeremiah Smithline	539 W. 52d street, New York City	3		do	do	Do.
	4	Joseph McPhilips	543 W. 52d street, New York City	2		do	do	Do.
	4	Patrick Ward	510 W. 53d street, New York City	3		do	do	Do.
	4	August Kampfine	530 W. 53d street, New York City	2		do	do	Do.
	4	Patrick Fox	615 W. 54th street, New York City	3		do	do	Do.
	4	Ann Wonderlich	631 W. 54th street, New York City	4		do	do	Do.
	4	Alexander Crooks	S side 37th below 11th avenue, New York City	12		Bad	Fair	Do.
	6	John Baden	SW. corner 64th and 10th avenue, New York City	1		Good	Good	Do.
	6	Michael Curry	W. side 64th, 13 door W. 10th avenue, New York City.	7		do	do	Do.
	6	Theodore Jacobi	W. side 64th, 8 door W. 10th avenue, New York City.	3		do	do	Do.
	6	Charlotte Simon	520 W. 61st street, New York City	1		do	do	Do.
	6	Henry Gier	W. side 64th st. 8 drs. w. 10th ave., New York City	1		do	do	Do.
	6	Amelia Decamp	NW. cor. 66th street and 11th ave., New York City	4		do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections in New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.							
Aug. 6	Henry Millingfeld	N. side 62d street and 10th avenue, New York City	1		Good	Good	C. B. Michener.
6	Kate McMann	N. side 62d street, 2 drs. f. 11th ave., New York City	5		do	do	Do.
6	C. Brown	435 W. 63d street, New York City	2		do	do	Do.
7	Union stock yards	60th street and North River, New York City			do	do	Do.
8	George Mitchell	67th street and 11th avenue, New York City	1		do	do	Do.
8	Daniel Thomas	SE. cor. 70th street and 12th avenue, New York City	8		do	do	Do.
8	Jas. Carroll	S. side 75th street, e. of Riverside, New York City	10		do	do	Do.
8	W. E. O'Brien	NW. corner 79th and 11th avenue, New York City	8		Very good	Very good	Do.
8	Mrs. West	W. s. 78th and e. of Riverside ave., New York City	1		Good	Good	Do.
8	John Miller	NE. cor. 80th, e. of Riverside ave., New York City	2		do	do	Do.
8	John Schwartz	503 W. 81st street, New York City	5		do	do	Do.
11	John D. Amean	W. s. W. 81st st., 3 h. w. Boulevard, New York City	1		do	do	Do.
11	Fred. Einberger	W. s. 81st street, 1 h. 11th avenue, New York City	2		do	do	Do.
11	Adolph Zerremer	W. s. 88th street, 1 h. e. 10th ave., New York City	1		do	do	Do.
11	Henry Haseman	SW. cor. 88th and e. 10th avenue, New York City	1		do	do	Do.
11	Fred. Schmidt	NW. cor. 88th and e. 10th avenue, New York City	1		do	do	Do.
12	Michael Murphy	S. s. 93d street, 1 h. w. 9th avenue, New York City	1		do	do	Do.
12	Rose McGloin	93d street between 8 and 9 avenues, New York City	1		do	do	Do.
12	John Brandeth	S. s. 93d st., 1 h. e. 11th avenue, New York City	2		do	do	Do.
12	Fred. Adler	92d street, 1 h. e. 11th avenue, New York City	1		do	do	Do.
12	Louis Wendell	92d street and 9th avenue, New York City	7		do	Very good	Do.
12	Frank Krall	97th street and 9th avenue, New York City	2		do	Good	Do.
12	Robt. Thompson	97th street and Western Boulevard, New York City	1		do	do	Do.
13	Wm. Wissen	S. s. 96th street, 1 h. to Boulevard, New York City	1		do	do	Do.
13	H. Guterding	S. s. 95th st., 1 h. to Boulevard, New York City	2		do	do	Do.
13	Wm. Daly	Riverside ave. bet. 96th and 97th sts., New York City	5		do	do	Do.
13	John Gruber	S. s. 96th st., 1 h. e. Boulevard ave., New York City	1		do	do	Do.
14	John Richardson	NE. cor. 107th st. and Boulevard ave., New York City	1		do	do	Do.
14	Mr. Kessel	S. s. 106th st., 1 h. e. Boulevard av., New York City	1		do	do	Do.
14	Peter Deil	108th st. bet. 10th and 11th aves., New York City	8		do	do	Do.
14	Michael Manning	SW. cor. 107th st. and 10th ave., New York City	3		do	do	Do.
14	Andrew Henderson	W. Boulevard bet. 104th and 105th sts., New York City	8		do	do	Do.
14	Mr. Marshall	104th st. and 9th ave., New York City	1		do	do	Do.
14	Henry Kruse	New ave. bet. 102d and 103d sts., New York City	1		do	do	Do.
14	Samuel McClintock	New ave. and 102d st., New York City	13		do	do	Do.
14	Henry Lange	N. s. 106th st., 1 h. w. 9th ave., New York City	2		do	do	Do.
14	John Fullman	S. s. 108th st., e. 10th ave., New York City	1		Fair	Fair	Do.
15	Union Stock Yards	60th st. and North River, New York City			Good	do	Do.
15	Margaret Bauer	S. s. 116th st., e. of 8th ave., New York City	1		do	Good	Do.

	15	Mrs. Kennely	106th st., 1 h. w. 8th ave., New York City	1		do	do	Do.
	15	Mrs. Whelan	106th st., 2 h. w. 8th ave., New York City	1		do	do	Do.
	15	Mrs. Elliott	106th st., 2 h. e. of 9th ave., New York City	2		do	do	Do.
	16	Henry Grinn	S. s. 110th st., 5 h. e. Boulevard, New York City	5		do	do	Do.
	16	Chris. Rader	do	1		do	do	Do.
	16	H. Stockhoff	SE. cor. 109th st. and W. Boulevard, New York City	1		do	do	Do.
	16	Adam Schaub	N. s. 110th st., 1 h. w. Boulevard, New York City	7		do	do	Do.
	16	Mr. Beck	109th st., 1 h. w. 10th ave., New York City	1		do	do	Do.
	16	J. Everett	109th st., bet. 10th ave. & W. Bo'vard, New York City	8		do	do	Do.
	16	Mr. Brumer	W. Boul'vard bet. 109th and 110th sts., New York City	12		do	do	Do.
	13	Mr. Brower	W. Boulevard, between 108 and 109, New York City	1		do	do	Do.
May	20	do	Pier 35	8		do	do	H. W. Rowland.
	20	do	Pier 34	35		do	do	Do.
	20	do	do	35		do	do	Do.
	27	do	do	14		do	do	Do.
June	3	do	Pier 35	8		do	do	Do.
	17	do	Pier 34	15		do	do	Do.
July	17	do	Pier 35	20		do	do	Do.
	17	do	Pier 34	19		do	do	Do.
	17	do	Pier 35	16		do	do	Do.
	22	do	do	19		do	do	Do.
	22	do	Pier 34. Franklin street, New York	10		do	do	Do.
Aug.	8	Sisters of Charity	Riverdale avenue, Mount Saint Vincent, New York	23		do	do	Do.
	8	F. C. Herriott	do	5		do	do	W. H. Wray.
	8	Edward Randolph	Pettner's lane, Mount Saint Vincent, New York	4		do	do	Do.
	8	Thos. H. Cuthbert	do	4		do	do	Do.
	9	Saml. Whitney	do	5		Poor	do	Do.
	9	Wm. Machelheim	do	1		Good	do	Do.
	9	Robert Martin	do	5		do	do	Do.
	9	Henry L. Stone	Palisade avenue, Riverdale, New York	2		do	do	Do.
	9	Sam'l Babcocks	do	5		do	do	Do.
	9	Robert Colgate	West street, Riverdale, New York	2		do	do	Do.
	9	W. H. Appleton	South street, Riverdale, New York	5		do	do	Do.
	9	Mrs. Ann Kennedy	Riverdale avenue, Mount Saint Vincent, New York	5		Poor	do	Do.
	11	Firman Lawrence	Jackson avenue, Spring road, Greenburg, New York	28		do	do	Do.
	11	Percy R. Pyne	Palisade avenue, Riverdale, New York	4		Good	do	Do.
	11	Henry Babcock	do	4		do	do	Do.
	12	Oliver Hariman	do	2		do	do	Do.
	12	Henry Spaulding	Spaulding Park, New York	1		do	do	Do.
	12	George Schemorhorn	Riverdale avenue, New York	1		do	do	Do.
	12	Wm. Thorne	do	1		do	do	Do.
	12	Thos. Thorne	do	1		do	do	Do.
	12	Waldo Hutchins	Riverdale avenue, Spuyten Duyvil, New York	8		do	do	Do.
	12	Stephen Crist	do	1		do	do	Do.
	12	Thos. Totten	do	1		do	do	Do.
	12	Michael Moran	do	2		do	do	Do.
	12	Patrick Kane	do	1		do	do	Do.
	13	Nathaniel Hall	Kensico, New York	6		Poor	do	Do.
	13	Fields Hall	do	21		Good	do	Do.
	14	James Norton	Riverdale avenue, Spuyten Duyvil, New York	2		do	do	Do.
	14	Mrs. Ewen	do	1		do	do	Do.
	14	Bowie Dash	Barney's lane, New York	5		do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections in New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.							
Aug. 14	Dennis Mulligan	Barney's lane, New York	6		Poor	Good	W. H. Wray.
14	James Douglas	Independent avenue, New York	2		Good	do	Do.
14	Henry Worthington	do	1		do	do	Do.
14	Albert Putnam	Spuyten Duyvil Hill, New York	1		do	do	Do.
14	Henry Smith	do	1		do	do	Do.
14	Isaac G. Johnson	do	2		do	do	Do.
14	J. R. Sergeant	do	2		do	do	Do.
14	Warren Sage	do	3		do	do	Do.
14	Patrick Gorman	Spuyten Duyvil road, New York	2		Poor	do	Do.
14	James Riley	do	2		do	do	Do.
14	Christ Martin	do	2		Good	do	Do.
15	John German	do	1		Poor	do	Do.
15	James Kennedy	Spuyten Duyvil Hill, New York	2		do	do	Do.
15	Hiram Barney	Cor. Riverdale ave. and Barney's lane, New York	1		Good	do	Do.
15	Mrs. Cary	Irvington, New York	1		do	Poor	Do.
15	Wm. Ward	Sprain Hill, Yonkers, New York	25		do	Good	Do.
16	Abram Odill	Sawmill river road, Yonkers, New York	32		do	do	Do.
16	John Mooney	do	6		Poor	do	Do.
18	Judson Jarvis	Riverdale lane, Riverdale, New York	8		Good	do	Do.
18	Patrick Magrudy	Sawmill river road, Yonkers, New York	9		Poor	do	Do.
18	G. P. Morrasini	Riverdale avenue, Yonkers, New York	1		Good	do	Do.
18	Robert Shepherd	do	1		do	do	Do.
18	J. G. Jarvis	Riverdale lane, Riverdale, New York	1		do	do	Do.
18	Joseph Bicknell	Albany post road, New York	1		do	do	Do.
18	J. Kilpatrick	do	1		do	do	Do.
18	Wm. H. Jones	do	6		Poor	do	Do.
18	Joseph Berrie	do	1		Good	do	Do.
19	John White	Broadway, near Yonkers, New York	17		Poor	do	Do.
19	J. W. Jones	do	1		Good	do	Do.
19	Isabella Murphy	Broadway, New York	2		Poor	do	Do.
19	Christian Keep	Rock street, New York	16		do	do	Do.
20	John Coleman	Mosholm avenue, New York	5		do	do	Do.
20	Walter Kelly	do	4		do	do	Do.
20	Benj. Odill	Sawmill river road, Greenburg, New York	2		Good	do	Do.
20	Jos. Denny	do	2		Poor	do	Do.
21	Mr. Ferris	Broadway, near Kingsbridge, New York	1		do	do	Do.
21	Dr. Wm. A. Varian	do	2		Good	do	Do.

21	Mr. Edwards	do	1		do	do	Do.
21	Jos. Godwin	do	3		do	do	Do.
21	George Mailer	do	1		do	do	Do.
21	Bernard Foy	do	2		do	do	Do.
21	Saml. Berrian	Church street, New York	1		do	do	Do.
21	Robert Murray	do	1		Poor	Good	Do.
21	Wm. Ackerman	do	1		Good	do	Do.
21	W. A. Smith	Albany avenue, New York	1		do	do	Do.
21	Jeremiah Healy	do	1		Poor	do	Do.
21	Mr. Faley	do	1		do	do	Do.
21	A. Shampnoy	Tuckahoe road, Yonkers, New York	22		Good	do	Do.
22	Mrs. Kelly	Church street, New York	2		Poor	do	Do.
22	Bernard Foy	do	2		do	do	Do.
23	Patrick Hooley	Between Hudson Park and River, New York	2		Good	do	Do.
23	Jeremiah Counell	Boston avenue, New York	4		do	do	Do.
23	Wm. Giles	do	1		do	do	Do.
23	Geo. Schrady	Albany avenue, New York	4		do	do	Do.
23	John Forbes	Broadway, New York	3		Poor	do	Do.
23	P. Malone	do	1		Good	do	Do.
23	Geo. Dorman	Dark Hill road, w. s. Boston avenue, New York	1		do	do	Do.
25	Geo. Livingston	Albany avenue and Bluff Park road, New York	27		Poor	do	Do.
25	Mrs. Martin	Albany avenue	2		do	do	Do.
25	Madge Kenney	Rock street, New York	2		do	do	Do.
25	J. M. Dykman	Broadway, New York	4		Good	do	Do.
25	Eugene Gibney	Broadway and 222d street, New York	1		do	do	Do.
26	Lawrence Drake	Broadway, New York	1		do	do	Do.
26	Wm. B. Isham	do	8		do	do	Do.
26	Terence Marion	do	2		do	do	Do.
27	Felix Kenan	do	5		Poor	do	Do.
27	Hugh Drenan	do	30		do	Dangerous	Do.
27	Matthew McQuade	do	4		do	Good	Do.
27	J. H. Gerkin	do	1		Good	do	Do.
27	August Schenck	do	1		do	do	Do.
27	William Keep	Beech street, New York	1		Poor	Good	Do.
27	Bridget Hamming	do	1		do	do	Do.
27	Catharine Moorley	do	1		do	do	Do.
28	Patrick Glennon	Riverdale avenue, New York	4		Good	do	Do.
28	John Margire	East and North River Railroad, between Spuyten Duyvil, New York.	3		Poor	do	Do.
28	John B. Cummings	Broadway, New York	1		Good	do	Do.
28	Mary Miller	do	1		do	do	Do.
28	John Corbit	Broadway and 207th street, New York	27	4	Poor	Dangerous	Do.
28	John White	Broadway and 208th street, New York	20		do	Good	Do.
28	James Devers	River road, Inwood, New York	1		Good	do	Do.
28	James McCrary	do	1		do	do	Do.
28	Dewit C. Hayes	Inwood Hill, Inwood, New York	1		do	do	Do.
28	Andrew Cormick	do	1		do	do	Do.
28	Joseph Keppler	do	1		do	do	Do.
28	Mr. Le Maier	Bailey avenue, New York	1		do	do	Do.
28	Christian Muller	do	1		Poor	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections in New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884. Aug. 18	John Cooper	W. s. 9th avenue, between 113th and 114th streets, New York.	5		Good	Good	C. B. Michener.
18	Henry Tone	E. s. 9th avenue, between 113th and 114th streets, New York.	8		do	do	Do.
18	Philip Kennedy	SW. cor. 10th avenue, between 113th and 114th streets, New York.	9		do	do	Do.
18	David Korn	112th street, between 10th avenue and Boulevard, New York.	7		do	do	Do.
18	Dr. Nichols	West'n Boulevard, between 116th and 117th streets, New York	33	7 c	Excellent	Very good	Do.
19	William Merchert	W. s. 121st street, 1st house e. Boulevard, New York.	11		Good	Good	Do.
19	Daniel Doyle	N. W. 121st street and Boulevard, New York	1		do	do	Do.
19	Jeremiah Lamb	W. side Boulevard, between 120th and 121st streets, New York.	1		do	do	Do.
19	Mrs. Banks	E. side Boulevard, between 120th and 121st streets, New York.	1		do	do	Do.
19	Michael Henn	1st h. w. near Boulevard, n. s. 121st street, New York	2		do	do	Do.
19	Henry Wagner	W. s. 10th avenue, between 121st and 122d streets, New York.	9		do	do	Do.
23	John Allen	Ninth avenue and 125th street, New York	10	2 a. c	do	do	Do.
23	Chris. Leuberger	S. s. 126th street and 9th avenue, New York	3		do	do	Do.
23	Dan'l E. Tieman	127th street and 12th avenue, New York	4		Very good	Very good	Do.
23	John Grimes	W. s. 129th street, near 9th avenue, New York	3		Good	Good	Do.
23	Conrad Haufling	W. s. 129th street, 2d h. e. 10th avenue, New York	1		do	do	Do.
23	Robt. Prier	E. s. Boulevard, bet. 125th and 126th sts., N. Y	2		do	do	Do.
23	Margaret Mangion	E. s. Boulevard, bet. 126th and 127th sts., N. Y	5		do	do	Do.
23	Mrs. James Grower	138th street, 2d h. w. 10th avenue, New York	1		do	do	Do.
23	Mr. J. Abbott	130th street, near 10th avenue, New York	2		do	do	Do.
23	Thos. O'Brien	126th street, near 10th avenue, New York	2		do	do	Do.
23	Ch. Schirarder	Between 95th and 96th streets, 8th avenue, New York	1		do	do	Do.
23	Mrs. Hoy	SW. corner 96th street, 8th avenue, New York	2		do	do	Do.
23	Michael McDermott	96th street, 1st h. w. 8th avenue, New York	2		do	do	Do.
23	Terence McDermott	127th street and 9th avenue, New York	12		do	do	Do.
25	Slaughter-houses	W. 11th avenue and 40th street, New York			do	do	Do.
25	Mr. Eastman	60th street and 11th avenue, New York			do	do	Do.
25	Union Stock Yards	60th street and North River, New York			do	do	Do.
26	William Zink	133d street and Saint Nicholas avenue, New York	1		do	do	Do.
26	August Wagner	141st street and Saint Nicholas avenue, New York	1		do	do	Do.
26	John Barthels	140th street and Saint Nicholas avenue, New York	3		do	do	Do.

	26	A. Klages	136th street and Saint Nicholas avenue, New York	1		do	do	Do.
	26	Henry Claussen	134th street, 1 h. e. 8th avenue, New York	2		do	do	Do.
	26	A. Wenzel	135th street and Saint Nicholas avenue, New York	2		do	do	Do.
	26	Herman H. Dirkes	133d street and Saint Nicholas avenue, New York	2		do	do	Do.
	26	Martin Sponlein	137th street and Saint Nicholas avenue, New York	2		do	do	Do.
	27	C. Koch	115th street and 8th avenue, New York	2		do	do	Do.
	27	James Malloy	N. s. 131st street, 1 h. e. 12th avenue, New York	6		do	do	Do.
	27	Eliza Mahon	N. s. 131st street, 1 h. w. Boulevard, New York	2		do	do	Do.
	27	John McCaffrey	SE. cor. 132d street and Broadway, New York	2		do	do	
	27	John Filer	S. s. 133d street, 1 h. e. Broadway, New York	1		Good	do	Do.
	27	John Dorst	do	5		do	do	Do.
	27	Matthew Conelly	SW. cor. 130th street and Broadway, New York	2		do	do	Do.
	27	Herman Juckman	S. s. 131th street, 2 h. e. 8th avenue, New York	1		do	do	Do.
	27	Edward Lawson	S. s. 134th street, 4 h. e. 8th avenue, New York	1		do	do	Do.
	30	Anthon Furst	N. s. 130th street, 1 h. e. 8th avenue, New York	1		do	do	Do.
	30	Fred. Kabilman	N. s. 139th street, east 8th avenue, New York	2		do	do	Do.
	30	Ernest Hurge	N. s. 137th street, 1 h. e. 7th avenue, New York	1		do	do	Do.
	30	Fred. Robker	S. s. 137th street, 1 h. e. 7th avenue, New York	1		do	do	Do.
	30	John Bartel	N. s. 141st street, 1 h. e. 8th avenue, New York	1		do	do	Do.
	30	John D. Cordes	N. s. 141st street, 2 h e. 8th avenue, New York	1		do	do	Do.
	30	Eliard Maixner	N. s. 142d street, 3 h. e. 8th avenue, New York	3		do	do	Do.
	30	Mrs. Mulcare	E. s. 148th street and 7th avenue, New York	12		do	do	Do.
	30	Thos. Watts	E. s. 141st street and 7th avenue, New York	1	1 c.	Very good	Very good	Do.
	30	Thos. McCovey	304 West 141st street, New York	1		Good	Good	Do.
	30	Geo. Losthill	305 West 141st street, New York	5		do	do	Do.
	29	P. S. Robinson	Albany post road, Van Courtlandt, New York	74		do	do	W. H. Wray.
	29	James Fay	do	2		do	do	Do.
	29	John Forsyth	do	1		Poor	do	Do.
	30	P. S. Robinson	do	27		Good	do	Do.
	30	Jos. S. Disbrow	Grand avenue, Woodlawn, New York	3		do	do	Do.
	30	Dr. Valentine	do	4		do	do	Do.
Sept.	1	John Carroll	Grand avenue, Van Courtlandt, New York	3		do	do	Do.
	1	M. Dooley	Jerome avenue, Woodlawn, New York	1		do	do	Do.
	1	J. Shanon	do	1		do	do	Do.
	1	Fred. Shrader	do	4		do	do	Do.
	1	L. Brosi	do	2		do	do	Do.
	1	Mr. Odell	Jerome avenue, Jerome Park, New York	1		do	do	Do.
	1	Thos. Webb	Jerome avenue, Woodlawn, New York	5		Poor	do	Do.
	1	Mr. Arnold	Sedgwick avenue, Kingsbridge, New York	1		Good	do	Do.
	1	Mr. Dunn	do	3		do	do	Do.
	1	H. B. Claflin	do	8		do	do	Do.
	1	Mrs. Clinchy	Kingsbridge road, New York	1		do	do	Do.
	1	Dennis Valentine	do	1		do	oo	Do.
	1	A. Weinisch	Gunn Hill road, Woodlawn, New York	3		do	do	Do.
	1	E. Weber	do	3		do	do	Do.
	1	Mr. Law	do	2		do	do	Do.
	1	Mrs Johnson	Gunn Hill road, Kingsbridge, New York	2		do	do	Do.
	1	Mr. Prince	do	3		Poor	do	Do.
	2	Mrs. Lappe	Gunn Hill road, Van Courtlandt, New York	1		Good	do	Do.
	2	P. Riley	do	1		do	do	Do.
	2	P. F. Bruno	do	3		do	do	Do.
	2	P. Roamer	Grand avenue, Woodlawn, New York	1		do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections in New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.							
Sept. 2	Cemetery Co	Grand avenue, Woodlawn, New York	6		Good	Good	W. H. Wray.
2	C. Cudlipp	Dickinson lane, Van Courtlandt New York	8		do	do	Do.
2	P. Austin,	Clinton avenue, Woodlawn, New York	2		do	do	Do.
2	E. Varian	Third street, Woodlawn, New York	1		do	do	Do.
2	Cor. McKay	Williamsbridge road, Williamsbridge, New York	1		do	do	Do.
2	Mr. Weeks	do	1		do	do	Do,
2	Mr. Dodge	do	1		do	do	Do.
2	Mr. McMara	do	1		Poor	do	Do.
2	Wm. Dissell	Mile Square, Woodlawn, New York	4		Good	do	Do.
2	Mr. Archer	do	3		do	do	Do.
2	Alex. Campbell	Masholm avenue, Woodlawn, New York	3		do	do	Do.
3	G. Metzger	Elm street, near Boulevard, New York	9	8	do	Dangerous	Do.
3	Julia E. Wremer	do	2		do	Good	Do.
2	Conrad Shafer	Arthur avenue, near Locust, New York	8		do	do	Do.
2	M. C. Connell	Arthur avenue, North 179th street, New York	6		Poor	do	Do.
2	E. Hiffeman	John street, New York	5		do	do	Do.
2	"The Brothers"	St. John's College, New York	21		Good	do	Do.
4	John B. Haskins	High Bridge road, Fordham, New York	4		do	do	Do.
4	Mrs. Briggs	Kingsbridge road, Fordham, New York	2		do	do	Do.
4	J. Dunn	2080 Vanderbilt avenue, New York	3		do	do	Do.
4	Sergeant Wright	Marion avenue, New York	1		do	do	Do.
4	Dr. Hedges	do	1		do	do	Do.
4	Thos. Evans	264 Marion avenue, New York	9		do	do	Do.
4	Jas. B. Thompson	S. Boulevard and R. R., Fordham, New York	1		do	do	Do.
4	Lawrence Kehoe	S. Boulevard, Webster avenue, Fordham, New York	2		do	do	Do.
4	Senator Miles	S. Boulevard and Boston road, Fordham, New York	2		do	do	Do.
4	Mrs Giblin	Berrian avenue, near St. John street, New York	1		do	do	Do.
4	Mrs. Lelchfield	Berrian avenue, Webster, New York	2		do	do	Do.
4	Dr. Eden	Williams's Bridge road, Ford, New York	1		do	do	Do.
4	Wm. Valentine	do	1		do	do	Do.
4	Judge Tapper	do	1		do	do	Do.
4	Wm. Briggs	do	3		do	do	Do.
4	C. Pebrie	179th street and Morris avenue, Fordham, N. Y	4		do	do	Do.
4	Mrs. Crane	177th street and Morris avenue, Fordham, N. Y	2		do	do	Do.
5	C. Miller	178th street and Morris avenue, Fordham, N. Y	2		do	do	Do.
5	Mrs. Geddes	179th street and Morris avenue, Fordham, N. Y	3		do	do	Do.
5	F. Kilpatrick	Kingsbridge road, Fordham, New York	1		do	do	Do.
5	John Trophagen	Avenue B, Fordham, New York	1		do	do	Do.

5	Mary Conlon	2275 Third street, Fordham, New York	1		Poor	do	Do.
5	Stephen Ray	183d street and Morris avenue, Fordham, N. Y	1		Good	Poor	Do.
5	John I. Huer	177th street and R. R., Fordham, New York	6		do	Good	Do.
5	Mr. Needham	188th street, near Morris avenue, New York	6		do	do	Do.
5	Richard Bruno	Valentine avenue, New York	8		do	do	Do.
5	O. S. Brndenburg	do	1		do	do	Do.
5	P. Ryan	do	8		do	do	Do.
5	Mrs. J. Murphy	183d street and River, New York	8		do	do	Do.
6	Mrs. Carr	181st street and River, New York	7		do	do	Do.
6	Mr. Tienan	River street, New York	10		Poor	do	Do.
6	Richard Berry	Tiebout avenue, New York	10		Good	do	Do.
6	Wm. Kennedy	Mile Square road, New York	2		do	do	Do.
6	John Kennedy	Grand avenue, near Aqueduct, New York	2		do	do	Do.
6	M. Redmond	Jerome avenue, near Washolm avenue, New York	28	3c	Filthy	Dangerous	Do.
8	Philip Berrian	High Bridge road, Fordham, New York	8		Good	Good	Do.
8	Mr. Bailey	1st street and avenue A, Fordham, New York	1		do	do	Do.
8	Mr. Wilson	Thomas avenue, Fordham, New York	8		Poor	do	Do.
8	Mr. Bazenke	do	2		Good	do	Do.
8	Mrs. Randall	2021 Fordham avenue, Tremont, New York	2		do	do	Do.
8	Mr. Faber	Fordham avenue, Fordham, New York	1		do	do	Do.
8	Mr. Klug	do	8		do	do	Do.
8	Mr. Whiten	Fordham depot, Fordham, New York	1		do	do	Do.
8	Mich. Larkin	2080 180th street, Fordham, New York	8		do	do	Do.
8	Nat. McKeeven	Vanderbilt avenue, Fordham, New York	1		do	do	Do.
8	Mrs. Burke	Near Vanderbilt avenue, Fordham New York	1		do	do	Do.
8	J. Gunn	Washington avenue, Fordham, New York	7		do	do	Do.
8	Mrs. Ryan	do	8		do	do	Do.
8	Mr. Burkhardt	Lorillard street, Fordham, New York	5		do	do	Do.
8	Mr. Mooney	Hoffman street, Fordham, New York	1		do	do	Do.
8	Mr. Dyer	Kingbridge road	1		do	do	Do.
8	Mrs. Coombs	do	1		do	do	Do.
8	McGuire	182d street, corner Madison avenue, F., N. Y	1		do	do	Do.
8	"Sisters of Charity"	Bayard street, Fordham, N. Y	1		do	do	Do.
1	Frank McGowan	S. s. 59th street, between 10th and 11th aves., N. Y	10		do	do	Do.
	Union Stock Yards	60th street and North River, N. Y		1a	do	do	C. B. Michener.
2	do	do					
8	H. M. Hood	S. s. 152d street, 2 h. n. w. St. Nicholas ave., N. Y	1		do	do	Do.
8	Ch. Curner	NW. corner 11th avenue and 155th street, N. Y	5		do	do	Do.
8	Alex. McGuire	SW. corner 10th avenue and 158th street, N. Y	2		do	do	Do.
8	Wm. Depperman	158th street, between 10th and 11th avenues, N. Y	3		do	do	Do.
8	Michael Mintz	157th street, 2d h. n. w., 10th avenue, N. Y	2		do	do	Do.
4	Wm. Wheelock	158th street, 2d h. n. w., 10th avenue, N. Y	2		do	do	Do.
4	John McKenna	157th street, between 10th and 11th avenues, N. Y	5		do	do	Do.
4	Shepherd T. Knapp	S. s. 158th street, between 11th and 12 avenues, N. Y	6		do	do	Do.
4	Mrs. Foster	158th street and 12th avenue, N. Y	1		do	do	Do
4	Mrs. Morse	161st street, between 11th and 12th avenues, N. Y	1		do	do	Do.
4	Carl Fring	157th street and St. Nicholas avenue, N. Y	2		do	do	Do.
6	John Corbet	207th street and Kingsbridge road, N. Y	82		do	do	Do.
22	Anthony Singer	147th street, w. St. Nicholas avenue, N. Y	4		do	do	Do.
22	Frank Koch	147th street, w. and St. Nicholas avenue, N. Y	8		do	do	Do.
22	Chris. Beetzer	151st street, w. 10th avenue, N. Y	1		do	Fair	Do.
22	F. C. Buck	do	2		do	Good	Do.

Investigation of contagious pleuro-pneumonia—Inspections in New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stables.	Condition of animals.	Name of inspector.
1884.							
Sept. 22	Henry Wernering	151st street, w. 10th avenue, New York	1		Good	Good	C. B. Michener.
22	J. Brown	147th street, w. Boulevard, N. Y	2		do	do	Do.
22	C. Bahrus	150th street, e. 11th avenue, N. Y	2		do	do	Do.
23	N. Feulner	do	1		do	do	Do.
23	M. O'Brien	147th street, w. 10th avenue, N. Y	9		do	do	Do.
23	Henry Eilerman	148th street, w. 10th avenue, N. Y	1		do	do	Do.
23	Mrs. Cunninghan	147th street, w. 10th avenue, N. Y	2		do	do	Do.
23	M. Cunningham	147th street, w. 10th avenue, New York	4		do	do	Do.
24	Wm. Guilfoile	144th street, w. 10th avenue, New York	2		do	do	Do.
24	Jas. Scanlan	147th street, w. Boulevard, New York	1		do	do	Do.
24	M. Ryan	145th street and 8th avenue, New York	2		do	do	Do.
24	H Bogart	145th street, bet. 7th and 8th avenue, New York	1		do	do	Do.
24	Dennis McMann	149th street, w. 8th avenue, New York	11		do	do	Do.
24	Gabriel Case	162d street and Central avenue, New York	3		do	do	Do.
24	John D. Barry	154th street and 7th, New York	2		do	do	Do.
24	Geo. Tiefel	122d street, Mt. Morris, New York	4		do	do	Do.
24	Conrad Gersoling	65th street, w. 10th avenue, New York	3		do	do	Do.
24	Chris. Wernert	do	8		do	do	Do.
24	Geo. Ratz	Bet. 65th and 66th sts., 10th avenue, New York	1		do	do	Do.
24	Philip Holz	64th and 65th sts., 10th avenue, New York	1		do	do	Do.
27	Henry Geyer	64th street, w. 10th avenue, New York	1		do	do	Do.
27	John Martin	10th avenue, bet. 64th and 65th sts., New York	1		do	do	Do.
27	Andrew Schalles	65th street, w. 10th avenue, New York	4		do	do	Do.
9	J. Missing	Taylor avenue, near Kingsbridge road, N. Y	12		do	do	W. H. Wray
9	Mr. Thurman	do	1		do	do	Do.
9	Mr. Gander	do	1		do	do	Do.
9	Mr. Wiegle	do	1		do	do	Do.
9	J. Huff	Jacob and Taylor ave., Kingsbride road, N Y	1		do	do	Do.
9	Mr. Wolf	Jacob and Taylor ave., Boulevard, New York	12		do	do	Do.
9	Mr. Leddy	Taylor ave., near Kingsbridge road, N. Y	1		do	do	Do.
9	Mr. Wilson	Garden, near Boulevard, New York	3		do	do	Do.
9	Geo. Jarman	do	1		do	do	Do.
9	Mr Engler	185th street, near Boulevard, New York	2		Poor	do	Do.
9	Mr. Cook	179th street, near Boulevard, New York	7		Good	do	Do.
9	Mr. Miller	183d street, near Boulevard, New York	7		do	do	Do.
10	Mr. Lowker	S. Boulevard, near Tremont avenue, New York	1		do	do	Do.
10	Mr. Gavon	K. B. road, near Boulevard, New York	2		do	do	Do.
10	P. Danbar	Orchard, near Chestnut, New York	2		do	do	Do.

10	Pat. Lunny	177th street, near West Farms, New York	17	do	do	Do.
10	Mrs. Gill	Orchard, near Chestnut, New York	1	do	do	Do.
10	D. Dugin	175th street and S. Boulevard, New York	10	do	do	Do.
10	William Nagle	Lyons street and S. Boulevard, New York	20	do	do	Do.
10	M. Shuh	1252 Woodruff avenue, New York	8	do	do	Do.
11	J Hohn	Woodruff avenue and S. Boulevard, New York	5	do	do	Do.
11	Mr. Beck	Boston avenue, south of S. Boulevard, New York	17	do	do	Do.
11	Mr. Jackson	do	2	do	do	Do.
11	Wm. Kengie	do	9	do	do	Do.
11	M. Underhill	do	3	do	do	Do.
11	Geo McFarlane	K. B. road, south of S. Boulevard, New York	1	do	do	Do.
12	Pat Green	K. B road, near Boston avenue, New York	52	do	do	Do.
12	Jas. Walker	Main street, bet. West ave. and P street, New York	6	do	do	Do.
12	Mrs. Tryon	do	8	do	do	Do.
13	Dr. Freeman	do	2	do	do	Do.
13	G. Shappell	176th street and Jerome avenue, New York	12	do	do	Do.
13	E. Vail	1819 Weeks street, New York	7	do	do	Do.
13	Thos. Hopkins	Corner Spring and Weeks street, New York	2	do	do	Do.
13	D Doyle	Morris avenue, bet. 173d and 174th street, New York	11	do	do	Do.
13	H. Ferrigan	1687 Washington avenue, New York	1	do	do	Do.
13	H. Shafer	170th street and Railroad avenue, New York	2	do	do	Do.
13	J. Sullivan	173d street and Railroad avenue, New York	1	do	do	Do.
13	Mr. Crawford	Orchard, near Jerome avenue, New York	1	do	do	Do.
16	J Stillwell	173d and Railroad avenue, New York	1	do	do	Do.
16	J. H. Weeks	do	1	do	do	Do.
16	P. Doyle	719 E. 171st street, New York	1	do	do	W. H. Way.
16	James Fitzpatrick	173d street, near Jerome avenue, New York	16	do	do	Do.
16	J. Knesemeyer	do	1	do	do	Do.
16	Mr. Dugan	Walnut, near 1st avenue, New York	3	do	do	Do.
16	Mr. Donohue	175th street, near R. R. avenue, New York	2	do	do	Do.
16	Mr. Cameron	K B. road and Jerome avenue, New York	2	do	do	Do.
16	Mr. Gauff	1797 Washington avenue, New York	1	do	do	Do.
16	Mr. Nixon	1734 Washington avenue, New York	2	do	do	Do.
16	Mr. Bonfields	1662 Washington avenue, New York	1	do	do	Do.
16	Mr. Arnold	1471 Washington avenue, New York	1	do	do	Do.
17	Mr. Diess	1429 Fordham avenue, New York	8	do	do	Do.
17	J. Prussa	1699 Fordham avenue, New York	2	do	do	Do.
18	Mr. Eaton	1761 Fordham avenue, New York	1	do	do	Do.
17	Mr. Heimberger	1849 Fordham avenue, New York	1	do	do	Do.
17	Mrs. Seple	Cor. 175th and Fordham avenue, New York	1	do	do	Do.
18	Mrs. Baile	1800 Madison avenue, Fordham, New York	1	do	do	Do.
18	Mrs. Doyle	1633 Bathgate avenue, New York	1	do	do	Do.
18	Mr F. Zeltner	170th street and Fordham avenue, New York	2	do	do	Do.
18	W. Neill	Franklin avenue, New York	9	Poor	do	Do.
18	J. McKown	Fairmount avenue and Broadway, New York	15	Good	do	Do.
18	Mr. Wilson	170th street and Boston avenue, New York	1	do	do	Do.
18	Wm Birrell	Boston avenue, near Broadway, New York	9	do	do	Do.
18	Mr. Janec	do	2	do	do	Do.
18	Mr. Handibo	170th street and Franklin avenue, New York	3	do	do	Do.
19	C. Bathgate	171st street and Boston avenue, New York	4	do	do	Do.
19	Mr. Beck	do	5	do	do	Do.
19	Mr. O'Brien	Bussing Lane, near Boston avenue, New York	30	do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections in New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stables.	Condition of animals.	Name of inspector.
1884.							
Sept. 19	M. Sherman	Catharine, corner Samuel street, New York	1		Good	Good	W. H. Wray.
19	Mr. Gaming	Catharine, cor. Samuel street, New York	1		do	do	Do.
19	Mr. Koch	Catharine and Center streets, New York	5		do	do	Do.
20	Mr. Trott	Boston avenue, near Jerome avenue, New York	14		do	do	Do.
20	Mr. Keller	Boston avenue, and Kingsbridge road, New York	2		do	do	Do.
20	Mr. Scofield	Maine, corner Kingsbridge road, New York	1		do	do	Do.
20	O. Tohen	Bayard and Cambreling avenue, New York	4		do	do	Do.
20	H. Hardmann	Woodruff avenue, near Prospect street, New York	1		do	do	Do.
20	Mr. Burkhardt	Lorillard and Kingsbridge road, New York	5		do	do	Do.
20	Mr. Welsh	Adams and Kingsbridge road, New York	3		do	do	Do.
22	E. Welsh	Kingsbridge road, New York	1		do	do	Do.
23	Mr. O. Donud	Main, near West avenue, New York	1		do	do	Do.
22	D. Mapes	do	1		do	do	Do.
22	Mr. Guffrey	West avenue, near Prospect street, New York	2		do	do	Do.
22	Mr. Mailey	Woodruff and Franklin avenue, New York	1		do	do	Do.
22	Mr. Edibol	Near Franklin avenue, New York	1		do	do	Do.
22	Mr. Hocking	Powell, near Fordham avenue, New York	3		do	do	Do.
22	Mr. Donnerein	2393 Lorillard street, New York	1		do	do	Do.
23	Mr. Poulsen	177th, near Railroad avenue, New York	1		do	do	Do.
23	Mr. Kirby	178th street, near Bathgate avenue, New York	1		do	do	Do.
23	Mr. McMahon	Adams, near Kingsbridge road, New York	1		do	do	Do.
23	Mr. Dunn	2080 Railroad avenue, New York	2		do	do	Do.
23	Mrs. Whalen	Adams, near Kingsbridge road, New York	1		do	do	Do.
23	J. Hanna	College, near Hoffman avenue, New York	1		do	do	Do.
23	Mrs. Connors	do	1		do	do	Do.
23	Mr. Randle	179th street and Fordham avenue, New York	1		do	do	Do.
24	A. Bane	White Plains road, White Plains, N. Y	80		do	do	Do.
24	W. Warren	do	8		do	do	Do.
24	Wm. Tompkins	do	2		do	do	Do.
24	John Gibson	White Plains road, White Plains, N. Y	6		do	do	Do.
25	P. Nolad	Jerome avenue, near Park, New York	4		do	do	Do.
25	Mr. Tooker	Elm, near S. Boulevard, New York	3		do	do	Do.
25	Mr. Murphy	Monroe, near Columbus avenue, New York	1		do	do	Do.
25	Mr. Mills	Taylor, near Kingsbridge road, New York	1		do	do	Do.
25	Mr. Donelly	do	1		do	do	Do.
25	Mr. Denny	do	1		do	do	Do.
25	Mr. Dalvin	Hoffman, near Kingsbridge road, New York	2		do	do	Do.
26	Mr. Hyland	Hoffman, near Jacob avenue, New York	1		do	do	Do.

	25	Mr. Mann	Jacob avenue and Kingsbridge road, New York	2		do	do	Do.
	25	Mr. Shandby	Jacob avenue and Arthur avenue, New York	2		do	do	Do.
	25	Mr. Tucemay	Arthur avenue and Pelham avenue, New York	1		do	do	Do.
	25	Mr. Hannahan	Hoffman and College, New York	2		do	do	Do.
	25	Mr. Daniel	do	2		do	do	Do.
	25	Mr. Bolster	Lorillard and S. Boulevard, New York	1		do	do	Do.
	29	Mr. Baker	Lorillard and Kingsbridge road, New York	3		do	do	Do.
	29	Jas. Malone	Kingsbridge road, near Adams street, New York	5	2	do	do	Do.
Oct.	1	J. B. Prote	North Broadway, Yonkers	3		do	do	Do.
	1	A. Curtice	do	1		do	do	Do.
	1	F. C. Havemeyer	do	1		do	do	Do.
	1	E. C. Moore	do	1		do	do	Do.
	1	W. W. Gage	do	1		do	do	Do.
	1	E. Weston	do	6		do	do	Do.
	1	W. F. Washburn	do	2		do	do	Do.
	1	R. W. Homes	do	1		do	do	Do.
	1	W. F. Cochran	do	7		do	do	Do.
	1	M. Nathan	do	2		do	do	Do.
	1	M. B. Copenthwaite	do	2		do	do	Do.
	1	E. A. Nichols	do	1		do	do	Do.
	1	Benj. Brown	do	7		do	do	Do.
	1	Hon. S. J. Tilden	do	14		do	do	Do.
	1	J. K. Myers	do	3		do	do	Do.
	1	C. H. Odell	do	1		do	do	Do.
	1	J. H Jaffray	do	1		do	do	Do.
	1	Jacob Hawley	do	1		do	do	Do.
	1	Weston	do	1		do	do	Do.
	1	C. H. Libinthal (est. of)	do	5		do	do	Do.
	1	G. L. Morse	do	3		do	do	Do.
	1	Mrs. J. H. Clark	do	2		do	do	Do.
	1	C. Martin (est. of)	do	1		do	do	Do.
	1	J. E. Andruss	do	11		do	do	Do.
	2	H. C. Bowers	Warburton avenue, Yonkers	1		do	do	Do.
	2	O. Stahlnecker	do	1		do	do	Do.
	2	O. Dudley	do	2		do	do	Do.
	2	Maj. F. Shannard	do	2		do	do	Do.
	2	B. Silliman	do	1		do	do	Do.
	2	R. C. Vilas	do	1		do	do	Do.
	2	John Nyland	Herriott street, Yonkers	6		do	do	Do.
	2	J. B. Colgate	Ravine avenue, Yonkers	2		do	do	Do.
	2	Mrs. Ford	Hawthorne avenue, Yonkers	1		do	do	Do.
	2	C. W. Cleveland	South Broadway, Yonkers	1		do	do	Do.
	2	Capt. J. Peene	Warburton avenue, Yonkers	1		do	do	Do.
	2	N. K. Shears	Ravine street, Yonkers	1		do	do	Do.
	2	John Clark	Hawthorne avenue, Yonkers	8		do	do	Do.
	2	Mrs. Weeks	Highland avenue, Yonkers	1		do	do	Do.
	2	B. E. Kingman	South Broadway, Yonkers	2		do	do	Do.
	2	James Bloomfield	South Broadway, Yonkers	2		do	do	Do.
	2	Joseph Peeve	Warburton avenue, Yonkers	1		do	do	Do.
	2	J. B. Trevor	do	6		do	do	Do.
	2	Daniel Hayes	Clinton street, Yonkers	8		do	do	Do.
	2	W. B. Edgar	Hawthorne avenue, Yonkers	2		do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections in New York—Continued.

Date.		Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Codition of animals.	Name of inspector.
1884.								
Oct.	2	Wich Larkin	Jefferson street, Yonkers	4		Good	Good	W. H. Wray.
	2	Mrs. Rollins	South Broadway, Yonkers	3		do	do	Do.
	3	Mrs. T. Radford	do	1		do	do	Do.
	3	R. P. Getty	do	2		do	do	Do.
	3	George Stewart	do	2		do	do	Do.
	3	Capt. J. Mangin	Post avenue, Yonkers	1		do	do	Do.
	3	J. H. Schieffelin	Riverdale avenue, Yonkers	4		do	do	Do.
	3	St. Aloysius Academy	South Broadway, Yonkers	1		do	do	Do.
	3	S. Lowene	do	2		do	do	Do.
	3	William Lawrence	do	1		do	do	Do.
	3	C. Kraft	do	1		do	do	Do.
	3	Joseph Proseus	Ludlow street, Yonkers	1		do	do	Do.
	3	B. F. Washburn	do	2		do	do	Do.
	3	Jewish Home	Riverdale avenue, Yonkers	2		do	do	Do.
	3	William Jones	do	1		do	do	Do.
	3	Capt H. G. H. Taw	South Broadway, Yonkers	1		do	do	Do.
	3	Mansion House estate	do	11		do	do	Do.
	2	D. Lyons	do	2		do	do	Do.
	3	James Pearce	do	1		do	do	Do.
	3	E. S. Johnson	Ludlow street, Yonkers	1		do	do	Do.
	3	George Purser	Riverdale avenue, Yonkers	2		do	do	Do.
	3	A. M. Dodge	do	2		do	do	Do.
	3	W. F. Nisbet	South Broadway, Yonkers	2		do	do	Do.
	3	S. Martin	do	1		do	do	Do.
	3	C. Daley	Herriott street, Yonkers	15		do	do	Do.
	4	Dennis Murray	McLean avenue, Yonkers	17		do	do	Do.
	4	Jas. Stewart	Woodworth avenue, Yonkers	2		do	do	Do.
	4	H. Austin	do	1		do	do	Do.
	4	S. B. Buick	do	1		do	do	Do.
	4	J. Wheeler	Locusthill avenue, Yonkers	2		do	do	Do.
	4	A. J. Alexander	do	1		do	do	Do.
	4	F. T. Holder	do	1		do	do	Do.
	4	C. Parsons	do	1		do	do	Do.
	4	G. Schwarb	McLean avenue, Yonkers	2		do	do	Do.
	4	J. McGuire	Glenwood avenue, Yonkers	1		do	do	Do.
	4	F. Debillier	Locusthill avenue, Yonkers	1		do	do	Do.
	4	Robt. Cowan	McLean avenue, Yonkers	11		do	do	Do.
	4	J. O'Neil	Woodworth avenue, Yonkers	1		do	do	Do.

4	E. Waving	Locusthill avenue, Yonkers	1		do	do	Do.
6	Ethan Flagg	Palisade avenue, Yonkers	2		do	do	Do.
6	Col. R. B. Blanchard	do	2		do	do	Do.
6	S. Halliday	do	1		do	do	Do.
6	W. H. Beers	do	1		do	do	Do.
6	Mrs. Currans	Garden street, Yonkers	3		do	do	Do.
6	J. Moynahan	do	1		do	do	Do.
6	Col. Hermance	Palisade avenue, Yonkers	1		do	do	Do.
6	Mr. Davidson	do	1		do	do	Do.
6	Chrs. Reed	South Broadway, Yonkers	1		do	do	Do.
6	Jacob Reed	Hefferhan avenue, Yonkers	1		do	do	Do.
6	C. Lockwood	Palisade avenue, Yonkers	1		do	do	Do.
6	J. C. Havemeyer	do	1		do	do	Do.
6	Geo. Reed	Waverly Place, Yonkers	1		do	do	Do.
6	Ned Walsh	Garden street, Yonkers	3		do	do	Do.
7	Mrs. Edwards	Lake avenue, Yonkers	30		do	do	Do.
7	Robt Kearns	do	6		do	do	Do.
7	John Cocklin	Orchard street, Yonkers	7		do	do	Do.
7	Wm. Grace	Hefferhan street, Yonkers	13		do	do	Do.
7	Jos. Palmer	do	13		do	do	Do.
7	Peter Prary	do	2		do	do	Do.
7	Jas. Caulfied	Lake avenue, Yonkers	2		do	do	Do.
7	Jos. Embree	Hefferhan street, Yonkers	1		do	do	Do.
8	H. R. Hicks	Tuckahoe road, Yonkers	2		do	do	Do.
8	A. Brawley	183 Gerome avenue, Yonkers	2		do	do	Do.
8	J McLaughlin	169 Gerome avenue, New York	1		do	do	Do.
8	Mrs. Finley	Bremen avenue, New York	7		do	do	Do.
8	Chrs. Dando	Kingsbridge road, New York	15		do	do	Do.
8	D. B. Henington	169 Gerome avenue, New York	2		do	do	Do.
8	J. H. Swan	do	1		do	do	Do.
8	Edward Mayer	Lind avenue, New York	2		do	do	Do.
8	Mr. Putnam	Spuyten Duyvil Hill, New York	1		do	do	Do.
8	Mich. Fish	169 Gerome avenue, New York	9		do	do	Do.
8	Peter Ward	Bremen avenue, New York	8		do	do	Do.
8	John Savor	Lind avenue, New York	4		do	do	Do.
8	C. Archer	Hefferhan avenue, New York	1		do	do	Do.
1	August Clegust	Between 133d and 134th sts. and St. N'las ave., N. Y.	1		do	do	C. B. Michener
1	Thos. Pindar	134th st. between 5th and 6th ave's, New York	4		do	do	Do.
1	F. M. Clark	146th st. and 8th ave., New York	1		do	do	Do.
1	Michael Barry	133d st., 5th h. w. 6th ave., New York	7		do	do	Do.
2 3 4	Union Stock Yards	60th st. and North River, New York			do	do	Do.
6	Henry Cleason	134th street, between 7th and 8th avenues, New York	8		do	do	Do.
6	J. Credan	158th street, 1 h. e. St. Nicholas avenue, New York	8		do	do	Do.
6	John Cooper	S. 160th street, between 10th and 11th avenues, New York.	1		do	do	Do.
6	James Mowles	W. corner 161st street and 10th avenue, New York	1		do	do	Do.
6	Mr. McMullins	South 161st street, near 11th avenue, New York	1		do	do	Do.
7	Nelson Chase	161st street, e. St. Nicholas avenue, New York	8		do	do	Do.
7	Robert Sauls	W. corner 162d street and 10th avenue, New York	1		do	do	Do.
7	Thos. Beatty	164th street, 1 h. e. 10th avenue, New York	2		do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections in New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884. Oct. 7	Mary Clancy	Between 165th and 166th streets, w. s. 10th avenue, New York.	10		Good	Good	C. B. Michener.
7	Bernard Murray	S. 165th street, 4 h. e. 10th avenue, New York	1		do	do	Do.
8	Edward Neff	153d street and 8th avenue, New York	2		do	do	Do.
8	Slaughter-house	40th street, w. of 11th avenue, New York			do	do	Do.
9	Union Stock Yards	60th street and North River, New York	4		do	do	Do.
10	Slaughter-house	40th street, w. of 11th avenue, New York			do	do	Do.
10	Chas. Engelbrecht	101st street and 10th avenue, New York	5		do	do	Do.
10	Mr. Laurence	104th street, 1 h. e. 9th avenue, New York	1		do	do	Do.
11	Chas. Schwarz	S. 174th street, 1 h. e. 10th avenue, New York	2		do	do	Do.
11	J. Hoodwright	S. 174th street, 1 h. w. Kingsbridge road, New York	3		do	do	Do.
11	John Barry	S. 175th street, 2d h. e. New avenue, New York	4		do	do	Do.
11	Wm. McDonald	S. 176th street, 1 h. w. 11th avenue, New York	2		do	do	Do.
11	Timothy Harrigan	W. corner 176th street and New avenue, New York	8		do	do	Do.
11	Edward Conway	175th street and Kingsbridge road, New York	1		do	do	Do.
11	Patrick Ward	N. 175th street, w. s. Kingsbridge road, New York	1		do	do	Do.
11	Mr. Bookman	do	2		do	do	Do.
13	Otto Brusen	S. Depot lane, 3 h. w. Kingsbridge road, New York	2		do	do	Do.
13	R. Rathbone	S. Depot lane, 1 h. w. Kingsbridge road, New York	3		do	do	Do.
13	Slaughter-houses	40th street, w. 11th avenue			do	do	Do.
14	Union Stock Yards	60th street and North River, New York			do	do	Do.
14	T. Eastman	59th street and 11th avenue, New York			Very good	Very good	Do.
15	John Holscher	SW. cor 113th street and St. Nicholas av., New York	2		do	do	Do.
15	H. Eihlerman	SW. cor. 114th street and St. Nicholas av., New York	1		do	do	Do.
15	F. Westercomb	S. 113th street, 2 h. w. St. Nicholas av., New York	1		do	do	Do.
15	Ph. Faulhaber	do	3		do	do	Do.
16	Union Stock Yards	60th street and North River, New York			do	do	Do.
16	Slaughter-houses	40th street, w. 11th avenue, New York			do	do	Do.
17	J. M. Hopkins	S. Depot lane, 5 h. w. Kingsbridge road, New York	2		do	do	Do.
17	D. Hollister	do	1		do	do	Do.
17	C. Tatam	W. cor. 174th st. and French Boulevard, New York	1		do	do	Do.
17	J. P. Martin	1 h. 174th street and French Boulevard, New York	2		do	do	Do.
17	F. F. Martin	W. cor. 174th street and French Boulevard, New York	2		do	do	Do.
17	Wm. Whiting	S. Depot lane, 1 h. e. N. R., New York	1		do	do	Do.
17	H. Perkins	W. cor. 177th street and Depot lane, New York	1		do	do	Do.
17	Mrs. McGuenn	1 h. n. 179th street, e. s. Kingsbridge road, New York	5		do	do	Do.
18	John Corbett	N. E. cor. 207th st. and Kingsbridge road, New York	32		do	do	Do.
18	Mary Miller	S. E. cor. 207th st. and Kingsbridge road, New York	1		do	do	Do.
18	Hugh Drennon	S. E. cor. 210th st. and Kingsbridge road, New York	30		do	do	Do.

	18	P. Keenon	do	5		do	do	Do.
	20	Jas. McCue	176th street, 1 h. e. 10th avenue, New York	3		do	do	Do.
	20	Mary J. Rodgers	E. cor. 182d street and Kingsbridge road, New York	1		do	do	Do.
	20	D. Pettigrew	E. cor. 184th street and 11th avenue, New York	2		do	do	Do.
	20	Mary Rooney	3 h. 184th street, w. s. 10th avenue, New York	2		do	do	Do.
	20	Wm. Broady	184th street and Kingsbridge road, New York	2		do	do	Do.
	20	John Dunn	E. cor. 187th street and Kingsbridge road, New York	8				Do.
	21	Union Stock Yards	60th street and North River, New York					Do.
	21	Slaughter-house						Do.
	23	F. Schrommer	W. cor. 190th street and 10th avenue, New York	2		Very good	Very good	Do.
	23	P. Clarus	192d street and 10th avenue, New York	3		Good	Good	Do.
	23	S. Kauper	192d street, 1 h. w. 10th avenue, New York	2		do	do	Do.
	23	M McCardle	do	3		do	do	Do.
	23	John Sowarby	193d street and Kingsbridge road, New York	2		do	do	Do.
	23	Patrick Kelly	195th street and Kingsbridge road, New York	1		do	do	Do.
	25	Union Stock Yards	60th street and North River, New York					Do.
	25	Harry Berthoff	100th street and 7th avenue, New York	4		Very good	Very good	Do.
	25	Slaughter-houses	40th street, w. 11th avenue, New York				do	Do.
Sept.	16	Barge Chas. Spear	Pier 34, North River, New York	9			do	Do.
	16	Steamboat City of Kingston	do	13			do	Thomas J. Herr.
	16	Steamer Ansonia	do	10			do	Do.
	18	J. Rosenberg	No. 327 Remington street, New York	5		Poor drainage	do	Do.
	16	Steamer Kaaterskill	Pier 35, North River, New York	40			do	Do.
	23	do	do	45			do	Do.
	23	Steamer Ansonia	Pier 34, North River, New York	14			do	Do.
	23	Steamer City of Kingston	Pier 35, North River, New York	8			do	Do.
	23	Barge Chas. Spear	Pier 34, North River, New York	8			do	Do.
	30	do	do	16		Poor drainage	do	Do.
Oct.	3	Fritz Opperman	329th street, E. 40th street, New York	1			do	Do.
Sept.	30	Steamer City of Kingston	Pier 35, North River, New York	4			do	Do.
	30	Steamer Ansonia	Pier 34, North River, New York	32			do	Do.
	30	Steamer Hasbrouck	do	4			do	Do.
	30	Steamer Redfield	do	10			do	Do.
	30	Steamer Kaaterskill	Pier 35, North River, New York	31			do	Do.
Oct.	6	Peter Dorlger	55th street and Avenue A, New York	1			do	Do.
	7	Steamer Hasbrouck	Pier 34, North River, New York	7			do	Do.
	7	Steamer Ansonia	do	6			do	Do.
	7	Steamer City of Kingston	Pier 35, North River, New York	2			do	Do.
	7	Barge Chas. Spear	Pier 34, North River, New York	8			Good	Do.
	7	Steamer Kaaterskill	Pier 35, North River, New York	44			do	Do.
	11	J. Hetch	No. 72 Delancey street, New York	6		Good	do	Do.
	11	Gotleib Slich	do	8		do	do	Do.
	14	Steamer Hasbrouck	Pier 35, North River, New York	4			do	Do.
	14	Steamer Ansonia	do	12			do	Do.
	14	Barge Chas. Spear	do	15			do	Do.
	14	Steamer Redfield	do	7			do	Do.
	14	Steamer City of Kingston	Pier 34, North River, New York	7			do	Do.
	14	Steamer Kaaterskill	do	33			do	Do.
	16	Moses Weil	2633 10th street, New York	1		Good	do	Do.
	17	F. Hannecker	133 Willets street, New York	2		do	do	Do.
	17	Pat. Ruddy	227 Delancey street, New York	1		do	do	Do.
	20	St. Joseph's C. A	89th street and Avenue A, New York	7			do	Do.

Investigation of contagious pleuro-pneumonia—inspections in New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.							
Oct. 20	Jacob Ruppert	91st street, between 1st and 2d avenues, New York	2			Good	Thomas J. Herr.
20	Picard	88th street, between 1st and 2d avenues, New York	2			do	Do.
20	Hunt	do	1			do	Do.
20	John Bumon	do	2			do	Do.
21	Steamer Redfield	Pier 35, North River, New York	4			do	Do.
21	Steamer Ansonia	do	1			do	Do.
21	Steamer Hasbronck	do	4			do	Do.
21	Barge Chas. Spear	do	7			do	Do.
21	Steamer City of Kingston	Pier 34, North River, New York	2			do	Do.
21	Steamer Kaaterskill	do	21			do	Do.
23	A. Crowhardt	710 E. 13th street, New York	1		Good	do	Do.
23	John Rhinefrank	132 Goerick street, New York	2			do	Do.
23	John Kent	128 Madison street, New York	1			do	Do.
23	Joe Lowell	402 E. 46th street, New York	2			do	Do.
9	H. B. Fuller	Tuckahoe road, Yonkers	1		Good	do	W. H. Wray.
9	Wm. Smith	do	8		do	do	Do.
9	J. C. Cowter	do	2		do	do	Do.
9	J. Murtha	do	3		do	do	Do.
9	M. Mooney	do	32		do	do	Do.
9	Mr. Daniell	do	1		do	do	Do.
9	Mrs. S. Fulton	do	2		do	do	Do.
10	E. L. Thomas	Yonkers avenue, Yonkers	1		do	do	Do.
10	John Moore	do	12		do	do	Do.
10	Mr. Trenchard	do	3		do	do	Do.
10	Christ O'Neil	Palmer avenue, Yonkers	11		do	do	Do.
10	Wm. Cronk	do	3		do	do	Do.
10	C. Weiderbold	Yonkers road, Yonkers	2		do	do	Do.
10	A. S. Odell	Tuckahoe road, Yonkers	1		do	do	Do.
10	Mr. Peckham	Yonkers avenue, Yonkers	7		do	do	Do.
10	M. L. Erhgott	do	2		do	do	Do.
10	Geo. Valentine	do	6		do	do	Do.
10	A. Grapanche	do	3		do	do	Do.
11	J. Buckhout	Sawmill river road, Ashford	2		do	do	Do.
11	R. J. Brown	Scarsdale road, Ashford	2		do	do	Do.
11	C. Lefurgy	do	11		do	do	Do.
11	Geo. Tompkins	do	8		do	do	Do.
11	A. Guyon	Sprain road, Ashford	3		do	do	Do.
11	A. Carterbien	Sawmill river road, Ashford	71		do	do	Do.

11	N. Smith	do	1		do	do	Do.
11	Capt. J. King	Scarsdale road, Ashford	5		do	do	Do.
11	D. D. Lefurgy	do	12		do	do	Do.
11	Miss Veile	Sprain road, Ashford	2		do	do	Do.
11	Geo. Lefurgy	Sawmill river road, Ashford	7		do	do	Do.
11	Geo. Jones	do	8		do	do	Do.
11	Mrs. Willsed	do	1		do	do	Do.
11	L. Lefurgy	Scarsdale road, Ashford	5		do	do	Do.
11	Wm. Acker	Sprain road, Ashford	15		do	do	Do.
11	J. Devoe	Jackson avenue, Ashford	5		do	do	Do.
13	Mrs. Hammond	Jackson avenue, Scarsdale	2		do	do	Do.
13	F. Gurling	Hartsdale road, Scarsdale	8		do	do	Do.
13	Geo. Sets	do	1		do	do	Do.
13	W. Jackson	Jackson avenue, Scarsdale	5		do	do	Do.
13	C. Parks	Ashford road, Scarsdale	5		do	do	Do.
13	H. Schilling	Hartsdale road, Scarsdale	7		do	do	Do.
13	Mr. Leviver	do	9		do	do	Do.
13	Geo. Suley	do	11		do	do	Do.
13	P. Kaufman	do	23		do	do	Do.
14	Isaac Odell	do	7		do	do	Do.
14	Geo. Smith	Sawmill river road, Scarsdale	2		do	do	Do.
14	J. Drissler	do	6		do	do	Do.
14	S. Willson	do	4		do	do	Do.
14	Cyrus W. Field	do	21		do	do	Do.
14	J. C. Horn	Dobbs' Ferry road, Scarsdale	1		do	do	Do.
14	John Wray	Sawmill river road, Scarsdale	2		do	do	Do.
14	D. B. Williams	do	2		do	do	Do.
14	Jas. Lynt	Hartsdale road, Ashford	5		do	do	Do.
14	Mrs. Horms	Dobbss Ferry road, Ashford	3		do	do	Do.
14	Geo. Frank	Sawmill river road, Ashford	1		do	do	Do.
14	H. R. Worthington	do	12		do	do	Do.
14	H. Tompkins	Hartsdale road, Ashford	3		do	do	Do.
14	C. Losee	Hartsdale road, Ashford	3		do	do	Do.
15	John Ackers	Sawmill river road, Elmsford	1		do	do	Do.
15	Thos. Allaire	Irvington road, Elmsford	6		do	do	Do.
15	Wm. Paton	Sawmill river road, Elmsford	5		do	do	Do.
15	H. Weller	Tarrytown road, Elmsford	1		do	do	Do.
15	A. Taxter	do	1		do	do	Do.
15	Mr. Amoux	do	1		do	do	Do.
15	N. Secor	Hartsdale road, Elmsford	4		do	do	Do.
15	J. Buckhout	Sawmill river road, Elmsford	3		do	do	Do.
15	Isaac Burr	Tarrytown road, Elmsford	2		do	do	Do.
15	Wm. Smith	do	4		do	do	Do.
15	H. Lander	Hartsdale road, Elmsford	11		do	do	Do.
15	Mr. Moyat	Sawmill river road, Elmsford	1		do	do	Do.
15	Dr. Minratt	Tarrytown road, Elmsford	2		do	do	Do.
16	Mr. Frazier	Broadway, Hastings.	2		do	do	Do.
16	Mrs. Hoppock	do	1		do	do	Do.
16	Isaac Lefurgy	do	12		do	do	Do.
16	E S. Mills	do	1		do	do	Do.
16	Mr. Brandt	do	1		do	do	Do.
16	Mrs. C. H. Talbot	do	1		do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections in New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884. Oct. 16	Mr. Mattison	Broadway, Hastings	2		Good	Good	W. H. Wray.
16	A. Kirkham	do	7		do	do	Do.
16	Mr. Christi	do	2		do	do	Do.
16	W. Rowley	do	4		do	do	Do.
16	Mrs. Hopke	do	2		do	do	Do.
16	M. L. Boutillier	do	4		do	do	Do.
16	Mrs. Minturn	do	3		do	do	Do.
16	W. K. Collins	do	2		do	do	Do.
17	H. V. Ambler	Broadway, Hastings	1		do	do	Do.
17	P. Amour	High street, Dobbs Ferry	1		do	do	Do.
17	J. W. Frazier	do	2		do	do	Do.
17	J. J. McComb	Broadway, Dobbs Ferry	2		do	do	Do.
17	C. Martin	Ashford avenue, Dobbs Ferry	1		do	do	Do.
17	Dr. Hasbrouck	Broadway, Hastings	1		do	do	Do.
17	Hon. D. O. Bradley	Broadway, Dobbs Ferry	2		do	do	Do.
17	P. M. Bergen	do	1		do	do	Do.
17	Thos. Paton	High street, Dobbs Ferry	2		do	do	Do.
17	F. W. Lasak	Ashford road, Dobbs Ferry	3		do	do	Do.
17	Dr. Judson	High street, Dobbs Ferry	1		do	do	Do.
17	Mr. Strange	Broadway, Hastings	1		do	do	Do.
17	E. Ebenspacher	Broadway, Dobbs Ferry	4		do	do	Do.
17	Henry Villard	High street, Dobbs Ferry	3		do	do	Do.
17	J. Storms	Broadway, Dobbs Ferry	4		do	do	Do.
18	W. Appleton	do	1		do	do	Do.
18	Jas. Wilds	do	4		do	do	Do.
18	D. A. Lindley	do	2		do	do	Do.
18	W. McClelland	Ashford avenue, Dobbs Ferry	1		do	do	Do.
18	Jas. Tyre	Broadway, Dobbs Ferry	2		do	do	Do.
18	J. Loan	do	1		do	do	Do.
18	J. Cottennett	do	2		do	do	Do.
18	F. J. Stone	Ashford avenue, Dobbs Ferry	2		do	do	Do.
18	M. Odell	Broadway, Dobbs Ferry	20		do	do	Do.
18	E. M. Field	do	2		do	do	Do.
20	A. Hamilton	do	4		do	do	Do.
20	J. Gibson	Broadway, Irvington	1		do	do	Do.
20	J. H. Whitehouse	do	3		do	do	Do.
20	Mr. Foster	do	1		do	do	Do.
20	H. H. Canon	do	2		do	do	Do.
20	F. W. Genteaq	do	3		do	do	Do.

20	T. Harper (estate)	do	2		do	do	Do.
20	J. Dibble	Irvington, Dobbs Ferry	4		do	do	Do.
20	J. McVickar	Broadway, Irvington	4		do	do	Do.
20	Geo. D. Morgan	do	7		do	do	Do.
20	E. S. Jaffray	do	9		do	do	Do.
20	A. S. Richards	do	1		do	do	Do.
20	H. R. P. Woodruff	do	4		do	do	Do.
21	Jay Gould	do	4		do	do	Do.
21	J. Mann	do	2		do	do	Do.
21	J. H. Ranker	do	8		do	do	Do.
21	J. Sullivan	Dublin road, Irvington	2		do	do	Do.
21	J. Taxter	do	1		do	do	Do.
21	A. Conklin	do	1		do	do	Do.
21	L. Buckbout	do	2		do	do	Do.
21	G. W. Smith	do	2		do	do	Do.
21	R. Skeel	Broadway, Irvington	2		do	do	Do.
21	H. R. Worthington	do	2		do	do	Do.
21	J. Crowese	do	2		do	do	Do.
21	J. Riley	Dublin road, Irvington	2		do	do	Do.
21	F. Washburn	do	1		do	do	Do.
21	A. Woods	do	2		do	do	Do.
22	Mr. Odell	Broadway, Tarrytown	2		do	do	Do.
22	J. Benedict	do	5		do	do	Do.
22	C. Reynolds	Sawmill river road, Tarrytown	5		do	do	Do.
22	R. M. Knapp	do	7		do	do	Do.
22	W. S. Gurvee	Broadway, Tarrytown	7		do	do	Do.
22	Mrs. Morse	do	2		do	do	Do.
22	C. Shelley	do	7		do	do	Do.
22	A. Decker	Sawmill river road, Tarrytown	14		do	do	Do.
23	J. H. Brown	do	18		do	do	Do.
23	J. W. Tompkins	do	10		do	do	Do.
23	R. Hoe (estate)	Broadway, Tarrytown	6		do	do	Do.
23	Stephen Brown	Sawmill river road, Tarrytown	22		do	do	Do.
23	B. Van Tassell	do	2		do	do	Do.
24	P. Hoe	Broadway, Tarrytown	8		do	do	Do.
24	J. Mott	do	2		do	do	Do.
24	T. H. Mead	do	2		do	do	Do.
24	J. Daniell	do	2		do	do	Do.
24	Capt. Cole	do	11		do	do	Do.
24	J. Dykeman	do	2		do	do	Do.
25	C. Sunderhill	Schoolhouse road, Tarrytown	11		do	do	Do.
25	W. Lawrence	Jackson avenue, Tuckahoe	5		do	do	Do.
25	C. Dusenberry	Schoolhouse road, Tuckahoe	13		do	do	Do.
25	B. Townsend	Jackson avenue, Tuckahoe	20		do	do	Do.
25	J. Underhill	Main road, Tuckahoe	8		do	do	Do.
25	S. Barker	Schoolhouse road, Tuckahoe	7		do	do	Do.
27	W. Smalley	Mamaroneck road, New Rochelle	19		do	do	Do.
27	Mr. Marley	Yonkers avenue, Yonkers	18		do	do	Do.
28	R. Merritt	Scarsdale road, Tuckahoe	9		do	do	Do.
28	J. Dusenberry	Near depot, Tuckahoe	7		do	do	Do.
28	S. Fee	do	4		do	do	Do.
28	J. Burwell	Yonkers road, Tuckahoe	4		do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections in New York—Continued.

Date.		Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884								
Oct.	28	R. White	Tuckahoe road, Tuckahoe	1		Good	Good	W. H. Wray.
	28	Mrs. D. Hodgeman	Scarsdale road, Tuckahoe	1		do	do	Do.
	28	J. Brown	do	4		do	do	Do.
	28	S. Leggett	do	3		do	do	Do.
	28	Dr. Nordquist	Lake road, Tuckahoe	1		do	do	Do.
	28	Steamer Kaaterskill	Pier 34, North River, New York City	38		do	do	Do.
	28	Steamer City Kingston	do	5		do	do	Do.
	28	Steamer Ansonia	Pier 35, North River, New York City	10		do	do	Do.
	28	Steamer Hasbrouck	do	5		do	do	Do.
	28	Steamer Redfield	do	8		do	do	Do.
	28	Barge Chas. Spear	do	12		do	do	Do.
	31	P. Maloney	222 E. 6th street, New York City	2		do	do	Do.
Nov.	4	Steamer Ansonia	Pier 35, North River, New York City	9		do	do	Do.
	5	Mrs. E. Hickman	636 E. 17th street, New York City	1		do	do	Do.
	5	William Ottman	237 E. 20th street, New York City	1		do	do	Do.
	5	William Odell	160 E. 23d street, New York City	1		do	do	Do.
	6	John Stillwell	214 E. 24th street, New York City	1		do	do	Do.
	6	Chas. Havman	215 E. 24th street, New York City	1		do	do	Do.
	6	Leop. Keller	do	1		do	do	Do.
	6	Jacob Dahlman	207 E. 24th street, New York City	1		do	do	Do.
	6	Isaac Dahlman	224 E. 24th street, New York City	1		do	do	Do.
	7	Sol. Mehrbock	152 E. 24th street, New York City	1		do	do	Do.
	7	Matthews	214 E. 24th street, New York City	1		do	do	Do.
	7	Chas. McCabe	204 E. 25th street, New York City	2		do	do	Do.
	7	John Harnett	17 E. 31st street, New York City	1		do	do	Do.
	8	Henry Ewer	223 E. 20th street, New York City			do	do	Do.
	8	John Dwyer	417 E. 22d street, New York City			do	do	Do.
	8	John Donnelly	10 E. 31st street, New York City			do	do	Do.
	8	Peter Dolinger	410 E. 53d street, New York City	1		do	Good	Do.
	8	Fritz. Opperman	329 E. 45th street, New York City	1		do	do	Thos. J. Herr.
	10	Marks	47th street, bet. 1st avenue and East River, N. Y. City	6		do	do	Do.
	10	Lerb Bros.	do	3		do	do	Do.
	10	Alfred Picard	58th street and East River, New York City	13		do	do	Do.
	10	Isaac Frank	49th street and East River, New York City	7		do	do	Do.
	10	John Kress	219 E. 54th street, New York City	2		do	do	Do.
	11	C. Brodrick	453 E. 54th street, New York City	8		do	do	Do.
	11	H. Eleas	409 E. 54th street, New York City			do	do	Do.
	11	Patrick Trophy	449 E. 54th street, New York City	13		Fair	do	Do.

11	Barge Chas. Speare	Pier 85 East River, New York City	10		do	do	Do.
11	Steamer Ansonia	do	12		do	do	Do.
11	Steamer Kaaterskill	Pier 84 North River, New York City	36		do	do	Do.
12	Geo. Schmidt	Cor. 90th street and 1st avenue, New York City	14		do	Fair	Do.
12	Mr. Englehardt	400 E. 92d street, New York City	8		Good	Good	Do.
12	St. Joseph's Orphan Asylum	Avenue A, corner 89th street, New York City	7		do	do	Do.
12	Conrad Dorr	467 E. 90th street, New York City	6		do	do	Do.
12	Geo. Ehret	2d avenue, bet. 91st and 92d streets, New York City	4		do	do	Do.
12	Chas. Reiger	203 E. 92d street, New York City	7		Poor	Fair	Do.
13	Michael Grogon	502 E. 71st street, New York City	22		do	do	Do.
13	M. Beck	508 E. 71st street, New York City	1		Good	Good	Do.
13	Pat. Shine	501 E. 71st street, New York City	1		do	do	Do.
13	Pat. Dean	68th street and East River, New York City	20		do	do	Do.
13	Aug. Braun	65th street and East River, New York City	5		do	do	Do.
14	Wm. Slack	77th street and East River, New York City	1		do	do	Do.
14	Bendeck Kurts	do	6		do	do	Do.
14	John H. Hickman	1435 Avenue A, New York City	3		do	do	Do.
14	Mary Furdein	423 E. 76th street, New York City	6		do	do	Do.
14	Mary Welch	435 E. 71st street, New York City	3		do	do	Do.
14	Montobeck	439 E. 71st street, New York	2		do	Fair	Do.
14	Miss Lennox	71st street and 5th avenue, New York	2		do	Good	Do.
14	S. M. Milliken	908 Madison avenue, New York	2		do	do	Do.
14	Ann Murphy	70th street and Madison avenue, New York	3		do	do	Do.
14	Henry Haley	78th street and Madison avenue, New York	2		do	do	Do.
15	John O'Connell	78th street and Madison avenue, New York	5		do	do	Do.
15	Dr. Nuddlebrook	172 E. 78th street, New York	3		do	do	Do.
8	Union Stock Yards	60th street and North River, New York			do	do	C. B. Michener.
8	T. Eastman	59th street and 11th avenue, New York			Very good.	Very good	Do.
8	Slaughter-houses	40th street west, and 11th avenue, New York			do	do	Do.
7	Union Stock Yards	68th street and North River, New York			do	do	Do.
11	do	do			do	do	Do.
15	do	do			do	do	Do.
4	J. Bell	Sawmill river road, Greenburg	3		do	do	W. H. Wray.
4	J. Grinen	do	6		do	do	Do.
4	E. Lent	Jackson avenue, Greenburgh	1		do	do	Do.
4	J. Molineaux	do	2		do	do	Do.
4	B. Haines	Farragut road, Greenburg	1		do	do	Do.
4	J. Hogan	North Broadway, Yonkers	2		do	do	Do.
4	W. Irvine	Pickle House road, Greenburg	9		do	do	Do.
6	W. Devitt	Depot road, Bronxville	9		do	do	Do.
6	C. Chambers	do	4		do	do	Do.
6	J. M. Masterton	do	3		do	do	Do.
6	A. Masterton	Tuckahoe road, Bronxville	2		do	do	Do.
6	F. Kraft	Midland avenue, Bronxville	1		do	do	Do.
6	J. Burke	White Plains road, Bronxville	13		do	do	Do.
8	J. Herriott	do	2		do	do	Do.
8	L. Dobbs	White Plains road, Scarsdale	8		do	do	Do.
8	L. Popham	do	5		Good	Good	Do.
8	W. Popham	do	7		do	do	Do.
8	J. Drake	do	5		do	do	Do.
8	C. H. Adams	Depot road, Scarsdale	4		do	do	Do.
8	W. J. Wheeler	Mamaroneck road, Scarsdale	2		do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections in New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.							
Nov. 8	E. J. Dunning	Mamaroneck road, Scarsdale	3		Good	Good	W. H. Wray.
10	J. Carpenter	do	17		do	do	Do.
10	Thomas Morrill	do	36		do	do	Do.
10	Estate of Dr. Bruen	do	4		do	do	Do.
10	H. Lefurgy	do	15		do	do	Do.
11	J. Butler	White Plains road, Hartsdale	3		do	do	Do.
11	J. Johnson	do	2		do	do	Do.
11	P. B. Odell	do	11		do	do	Do.
11	W. H. Archer	do	16		do	do	Do.
11	E. J. Quimby	do	7		do	do	Do.
11	J. Sweet	do	2		do	do	Do.
11	J. Reed	do	5		do	do	Do.
11	W. Swan	do	8		do	do	Do.
11	H. Drury	do	1		do	do	Do.
11	H. Bates	do	18		do	do	Do.
12	A. E. Reynolds	White Plains road, Scarsdale	23		do	do	Do.
12	D. A. Weed	do	2		do	do	Do.
12	George Ferris	do	6		do	do	Do.
12	O. Hyatt	do	9		do	do	Do.
13	R. Dusenberry	Ashford road, Scarsdale	11		do	do	Do.
13	Christ. Moore	Yonkers avenue, Yonkers	15		do	do	Do.
13	S. Valentine	Ashford road, Hartsdale	14		do	do	Do.
13	W. Wright	do	32		do	do	Do.
13	Dykman Odell	do	12		do	do	Do.
14	H. Wright	do	26		do	do	Do.
14	W. Hawlett	do	17		do	do	Do.
14	Stephen Hopkins	do	13		do	do	Do.
14	Frank Lawrence	do	6		do	do	Do.
15	P. Kaufman	Ashford avenue, Hartsdale	46		do	do	Do.
15	J. O'Brien	Elmsford road, Hartsdale	1		do	do	Do.
15	F. A. Decker	Sawmill River, Tarrytown	27		do	do	Do.
15	J. Eppes	Elmsford, Hartsdale	2		do	do	Do.
3	Slaughter-houses	47th street and 1st avenue, New York	1		do	do	Thos. J. Herr.
4	do	do			do	do	Do.
5	do	do	2	1	do	do	Do.
6	do	1st avenue, between 45th and 46th streets, New York	3		do	do	Do.
6	do	47th street and 1st avenue, New York	1		do	do	Do.
6	do	45th street and 1st avenue, New York	5		do	do	Do.
7	do	1st avenue, between 45th and 46th streets, New York	19		do	do	Do.

	7	do	do	10	2	do	do	Do.
	8	do	47th street and 1st avenue, New York	8		do	do	Do.
Oct.	27	do	do			do	do	Do.
	27	do	45th street and 1st avenue, New York	2		do	do	Do.
	28	do	47th street and 1st avenue, New York	5		do	do	Do.
	28	do	do	1		do	do	Do.
	28	do	1st avenue between 46th and 47th streets, New York	4		do	do	Do.
	29	do	47th street and 1st avenue, New York	3		do	do	Do.
	30	do	do	6	1	do	do	Do.
	31	do	do	1		do	do	Do.
Nov.	1	do	do			do	do	Do.
	3	do	At the dock, New York			do	do	Do.
Nov.	4	Slaughter-houses	do			do	do	Do.
	5	do	Pier foot Maiden lane, New York	1		do	do	Do.
	6	do	90th street, between 1st and 2d avenues, New York	1		do	do	Do.
	7	do	At the dock, New York			do	do	Do.
	8	do	Morris avenue, New York	1		do	do	Do.
Oct.	27	do	113th street and 5th avenue, New York	1		do	do	Do.
	27	do	207th street and Kingsbridge road, New York	1		do	do	Do.
	28	do	60th street stock yard, New York	1		do	do	Do.
	28	Offal dock	510 E. 14th street, New York	1		do	do	Do.
	29	do	At the dock, New York	1		do	do	Do.
	30	do	do	1		do	do	Do.
	31	do	do			do	do	Do.
Nov.	1	do	do			do	do	Do.
	10	Slaughter-house	45th street, near 1st avenue, New York	1		do	do	Do.
	10	do	47th street, near 1st avenue, New York	2	1	do	do	Do.
	10	do	1st avenue, between 46th and 47th streets, New York	14		do	do	Do.
	11	do	do	13		do	do	Do.
	12	do	45th street, near 1st avenue, New York	3		do	do	Do.
	12	do	47th street, near 1st avenue, New York	6		do	do	Do.
	13	do	do	6	3	do	do	Do.
	13	do	1st avenue, between 46th and 47th streets, New York	6		do	do	Do.
	14	do	47th street, near 1st avenue, New York	1		do	do	Do.
	14	do	45th street, near 1st avenue, New York	1		do	do	Do.
	14	do	1st avenue, between 46th and 47th streets, New York	9		do	do	Do.
	15	do	do			do	do	Do.
	10	Offal dock	60th street stock-yard, New York	1		do	do	Do.
	11	do	91st street and 1st avenue, New York	1		do	do	Do.
	12	do	On the dock, New York			do	do	Do.
	13	do	98th street and 2d avenue, New York	1				Do.
	14	do	On the dock, New York					Do.
	15	do	do					Do.
	17	J. M. Fisher	999 5th ave., New York			Good		Do.
	17	S. M. Smith	50 E. 62d street, New York			do		Do.
	17	S. M. Hamilton	107 E. 62d street, New York			do		Do.
	17	Mrs. M. Lynch	89th st., bet. 5th and Madison avenues, New York	20		do	Good	Do.
	17	John O'Connor	do	19		do	do	Do.
	17	Gertrude Falk	78th and 79th streets, Broadway av., New York	14		do	do	Do.
	17	H. Ohman	70th street and Boulevard, New York	2		do	do	C. B. Michenor.
	18	Union Stock Yards	60th street and North River, New York				Fair	Do.
	18	Steamer Redfield	Pier 35, North River, New York	6			Good	Thos. J. Herr.

Investigation of contagious pleuro-pneumonia—Inspections in New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1894.							
Nov. 18	Steamer Hasbrouck	Pier 35, North River, New York	4			Good	Thos. J. Herr.
18	Steamer Ansonia	do	12			do	Do.
18	Barge Charles Spear	do	11			do	Do.
18	Steamer Kaaterskill	Pier 34, North River, New York	35			do	Do.
18	Steamer City of Kingston	do	4			do	Do.
18	Netis	5th avenue between 77th and 78th streets, New York	2			do	Do.
20	John Nealon	99th street near 2d avenue, New York	4		Fair	Fair	Do.
20	Steve. Reilly	58 E. 89th street, New York	9		Good	Good	Do.
20	John Coleman	60 E. 89th street, New York	11		do	do	Do.
20	John McDonough	S. Croton st. 2d h. east K'sbridge road, New York	12		do	do	C. B. Michener.
20	H. Berthoff	116th street and 7th avenue, New York	4		do	de	Do.
21	Wm. Callehan	168th street E. Kingsbridge, New York	1		do	do	Do.
21	Phillip Blatner	165th street and East Kingsbridge, New York	1		do	do	Do.
21	Godfrey Snyder	S. 168th st., one house west 10th avenue, New York	1		do	do	Do.
21	Susan Healey	S. Croton st., three houses west 10th ave., New York	1		do	do	Do.
21	John Burns	98th street and East River, New York	8		do	do	Thos. J. Herr.
21	Thos. Gavin	109th street and East River, New York	19		do	do	Do.
21	John McDermott	347 99th street, New York	17		do	do	Do.
22	A. Crownhardt	710 E. 13th street, New York	1		do	do	Do.
22	F. Hanacker	183 Willets street, New York	2		do	do	Do.
22	Mrs E. Hickman	635 E. 17th street, New York	1		do	do	Do.
22	Jacob Dalhman	207 E 24th street, New York	1		do	do	Do.
24	Chas. Rieger	203 E. 92d street, New York	7		do	do	Do.
24	Geo. Schmidt	90th street and 1st avenue, New York	14	2	do	Fair	Do.
24	Mic. Englehardt	92d street near 1st avenue, New York	3		do	do	Do.
24	Conrad Dorr	407 E 90th street, New York	8		do	Good	Do.
24	Geo. Schmidt	90th street and 1st avenue, New York	14	2	do	Fair	Do.
24	Sol. Mehback	152 E 24th street, New York	1		do	Good	Do.
25	Steamer Hasbronck	Pier 35, North River, New York	7			do	Do.
25	Steamer Ansonia	do	15			do	Do.
25	Steamer Redfield	do	8			do	Do.
25	Barge Chas. Spear	do	7			do	Do.
25	Steamer City of Kingston	Pier 34 North River, New York	4			do	Do.
25	Steamer Catskill	do	63			do	Do.
25	H. Nauman	321 E. 60th street, New York	1		Good	do	Do.
25	John Long	408 E. 63d street, New York	3		do	do	Do.
25	D. Ko n	112th street, near Boulevard, New York	7		do	do	C. B. Michener.
25	Dr. Nichols	116th and 117th streets, near Boulevard, New York	33	3	Very good	Very good	Do.
26	Union Stock Yards	60th street and North River, New York					Do.

26	Aug. Shehan	317 E. 63d street, New York	9		Good	Good	Thos. J. Herr.
26	D. Sanders	325 E. 63d street, New York	1		do	do	Do.
26	Jerry Healey	73d street and East River, New York	1		Fair	do	Do.
26	Albert Rutloff	73d street between Avenues A and B, New York	1		Good	do	Do.
26	H. Nigmore	do	2		do	do	Do.
27	William Erath	73d street, between Avenues A and B, New York	3		do	do	Do.
27	Michael Gleason	do	3		do	do	Do.
27	Mrs. E. Mahr	do	7		do	do	Do.
27	William Gordon	502 71st street, New York	11		do	do	Do.
27	George Tieful	122d street and Mt. Morris avenue, New York	4		Excellent	do	C. B. Michener.
27	John Kills	E. corner 197th street and K'bridge road, New York	2		Good	do	Do.
27	Danl. Corkery	197th street, east Kingsbridge road, New York	2		do	do	Do.
27	Mrs. J. McDonald	do	1		do	do	T. J. Herr.
27	Mrs. Maggie Welch	435 E. 71st street, New York	3		do	do	Do.
28	Mrs. E. McCabe	72d street and East River, New York	4		do	do	Do.
28	Mrs. E. Matthews	75th street and East River, New York	1		do	do	Do.
28	Vincent Geisler	74th street and East River, New York	1		do	do	Do.
28	James McGennis	446 E. 75th street, New York	6		do	do	Do.
28	R. C. Combes	E. corner 175th street and 10th avenue, New York	2		do	do	Do.
28	Hugh Fletcher	S. 78th street, 1 h. w. 10th avenue, New York	2		do	do	Do.
28	M. Mathew	E. corner 210th and Cooper streets, New York	4		do	do	Do.
28	T. Morron	E. cor. Cooper street and Emerson ave., New York	2		do	do	Do.
29	H. Haese	105th street, near 1st avenue, New York	8	1 c.	do	do	Do.
29	Aremeili	107th street, bet. 1st street and Ave. A, New York	9	2 c.	do	Fair	Do.
29	Geo. Carage	do	7		do	Good	Do.
20	Mrs. A. Fuhr	102d street and 2d avenue, New York	2		do	do	Do.
29	Mrs. Holoran	4th avenue, between 75th and 76th sts., New York	3		do	do	Do.
29	Briggs & Taylor	225th street and Kingsbridge road, New York	1		do	do	C. B. Michener.
29	E. Gibney	224th street and Kingsbridge road, New York	1		Very good	Very good	Do.
29	Johnes Forbes	220th street and Kingsbridge road, New York	3		Good	Good	Do.
29	J. M. Dykman	218th street and Kingsbridge road, New York	4		do	do	Do.
29	Wm. B. Ishiam	212th street and Kingsbridge road, New York	6		do	do	Do.
29	Henry Gerkin	210th and Cooper streets, New York	1		do	do	Do.
29	John B. White	213th st., and Kingsbridge road, New York	16		do	do	Do.
17	D. O. Archer	Broadway, Tarrytown	3		do	do	W. H. Wray.
17	J. Pritchard	do	4		do	do	Do.
17	A. E. Orr	do	1		do	do	Do.
17	A. H. Jocelyn	do	2		do	do	Do.
17	C. K. Buchanen	do	1		do	do	Do.
17	Geo. Bechtel	do	5		do	do	Do.
17	P. Conrad	Irvington road, Irvington	3		do	do	Do.
17	Mott Jacob	Broadway, Tarrytown	1		do	do	Do.
17	H. G. Gilbert	do	1		do	do	Do.
17	E. J. Blake	do	2		do	do	Do.
17	J. Lee	do	16		do	do	Do.
17	N. H. Odell	do	2		do	do	Do.
18	G. G. Stevens	do	24		do	do	Do.
18	Geo. Daniel	Prospect street, Tarrytown	2		do	do	Do.
18	Jos. Young	Broadway, Tarrytown	2		do	do	Do.
18	W. Wallace	do	4		do	do	Do.
18	J. F. Kendall	Beekman avenue, Tarrytown	2		do	do	Do.
18	Geo. J. B. Brown	do	1		do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections in New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.							
Nov. 19	Cr. Rockwell	Benedict avenue, Tarrytown	2		Good	Good	W. H. Wray.
19	Jordan Mott	Cottage Place, Tarrytown	1		do	do	Do.
19	C. H. Curvier	McKee avenue, Tarrytown	1		do	do	Do.
19	W. E. Dodge	Prospect street, Tarrytown	8		do	do	Do.
19	Geo. Roberts	do	2		do	do	Do.
19	A. C. Kingsland	Kingsland Point, Tarrytown	5		do	do	Do.
19	W. H. Hoyt	Prospect street, Tarrytown	8		do	do	Do.
19	W. D. Humfries	Main street, Tarrytown	1		do	do	Do.
19	Wm. King	College street, Tarrytown	1		do	do	Do.
19	E. Mabie	Washington avenue, New York	2		do	do	Do.
20	H. W. Arthur	Sparta road, Scarborough	7		do	do	Do.
20	L. B. Wright	Broadway, Scarborough	3		do	do	Do.
20	Jacob H. Vantassel	Sleepy Hollow road, Scarborough	18		do	do	Do.
20	L. B. Schieffelin	Sparta road, Scarborough	8		do	do	Do.
20	J. E. Williams	do	4		do	do	Do.
20	G. L. Aspinwall	Broadway, Scarborough	17		do	do	Do.
20	J. W. Mason	do	5		do	do	Do.
20	James H. Vantassel	Broadway, Sleepy Hollow	32		do	do	Do.
21	C. Anderson	Croton street, Sing Sing	18		do	do	Do.
21	H. Brewster	do	29		do	do	Do.
21	N. H. Baker	152 Main street, Sing Sing	2		do	do	Do.
21	R. Williams	119 Main street, Sing Sing	1		do	do	Do.
21	J. Sutton	Croton avenue, Sing Sing	1		do	do	Do.
21	J. B. Nexon	do	1		do	do	Do.
21	H. C. Nelson	161 Main street, Sing Sing	1		do	do	Do.
21	C. D. Rice	Croton avenue, Sing Sing	2		do	do	Do.
21	R. Smith	do	17		do	do	Do.
22	F. Larkin	Highland avenue, Sing Sing	1		do	do	Do.
22	J. P. Henop	do	1		do	do	Do.
22	D. D. Mangin	do	4		do	do	Do.
22	C. O. Joline	do	1		do	do	Do.
22	W. A. Brown	do	1		do	do	Do.
22	C. Brandreth	do	5		do	do	Do.
22	W. Stewart	do	2		do	do	Do.
22	J. Stern	do	1		do	do	Do.
22	J. Collard	134 Spring street, Sing Sing	16		do	do	Do.
23	J. Gibson	Eastern, Sing Sing	4		do	do	Do.
23	R. Williams	119 Main street, Sing Sing	1		do	do	Do.
23	C. French	88 Main street, Sing Sing	9		do	do	Do.

	23	E Ellis	Aqueduct street, Sing Sing	22		do	do	Do.
	23	J. C. Hyatt	Madison avenue, Sing Sing	6		do	do	Do.
	23	J. Hyland	Mill street, Sing Sing	18		do	do	Do.
	24	J. Klunder	N. Malcolm street, Sing Sing	8		do	do	Do.
	24	A. Wright	Aqueduct street, Sing Sing	1		do	do	Do.
	24	W. Sarles	Everett avenue, Sing Sing	42		do	do	Do.
	24	E. A. McAlpine	N. Malcolm street, Sing Sing	8		do	do	Do.
	25	E. A. Holbrook	Sparta road, Sing Sing	1		do	do	Do.
	25	J. Walsh	Broadway, Sing Sing	4		do	do	Do.
	25	J. Hand	do	19		do	do	Do.
	25	C. C. Clarke	Sparta road, Sing Sing	8		do	do	Do.
	25	S. B. Meade	Broadway, Sing Sing	32		do	do	Do.
	26	A. J. Tompkins	do	2		do	do	Do.
	26	W. Williams	Lafayette avenue, Sing Sing	1		do	do	Do.
	26	J. H. Townsend	William street, Sing Sing	7		do	do	Do.
	26	J. H. Underhill	Clinton avenue, Sing Sing	1		do	do	Do.
	26	Jos. Shute	Hill street, Sing Sing	1		do	do	Do.
	26	G. E. Smith	37 Broad street, Sing Sing	8		do	do	Do.
	26	J. L. Wheeler	Briar Cliff, Sing Sing	8		do	do	Do.
	26	F. Young	14 Mott street, Sing Sing	2		do	do	Do.
	28	V. Altman	White Plains road, Mount Vernon	2		do	do	Do.
	28	S. Busteed	do	7		do	do	Do.
	28	E. S. Barker	Eastchester road, Mount Vernon	4		do	do	Do.
	28	P. Jordan	do	5		do	do	Do.
	28	P. Harvey	do	4		do	do	Do.
	28	J. J. Armour	do	8		do	do	Do.
	28	J. A. Price	do	12		do	do	Do.
	28	F. H. Hunt	do	7		do	do	Do.
	28	J. Davis	do	2		do	do	Do.
	28	V. Hoog	White Plains road, Mount Vernon	1		do	do	Do.
	28	W. A. Roberts	do	7		do	do	Do.
	28	O. Bertine	do	1		do	do	Do.
	28	J. Thurston	Yonkers avenue road, Mount Vernon	5		do	do	Do.
	28	A. Pease	do	2		do	do	Do.
	29	J. Hacombe	44 9th avenue, Mount Vernon	2		do	do	Do.
	29	C. A. Meritt	Chester Hill, Mount Vernon	4		do	do	Do.
	29	C. Craney	do	2		do	do	Do.
Dec.	1	Henry Konig	S. 36th street and 1st avenue, New York	8		do	do	T. J. Herr.
	1	John Ryan	512 E. 82d street, New York	2		do	do	Do.
	1	C. Hurge	82d street and Avenue B, New York	1		do	do	Do.
	2	Mrs. Ann Murphy	5th avenue, between 75th and 76th street, New York	8		do	do	Do.
	2	City of Kingston	Pier 34 North River, New York	1		do	do	Do.
	2	Steamer Catskill	do	64		do	do	Do.
	2	Steamer Ansonia	Pier 35 North River, New York	12		do	do	Do.
	2	Steamer Hasbrouck	do	1		do	do	Do.
	2	Barge Chas. Spear	do	1		do	do	Do.
	2	Steamer Redfield	do	6		do	do	Do.
	3	S. Frier	526 E. 84th street, New York	1		do	do	Do.
	3	H Merz	525 E. 84th street, New York	2		do	do	Do.
	3	Wm. Nelson	84th street and E. River, New York	1		do	do	Do.
	4	Mrs. Muller	321 E. 103d street, New York	8		do	do	Do.
	4	Harry Leonard	319 103d street, New York	2		do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections in New York—Continued.

Date.		Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.								
Dec.	4	Geo. Florshite	E. 103d street, New York	6		Good	Good	T. J. Herr.
	4	John Distela	103d street and 2d avenue, New York	8		do	do	Do.
	5	F. Baumfield	103d street and 1st avenue, New York	5		do	do	Do.
	5	John Hempling	323 103d street, New York	1		do	do	Do.
	8	John Coleman	60 E. 89th street, New York	11		do	do	Do.
	8	Dennis Murphy	92d street and 5th avenue, New York	23		do	do	Do.
	9	Steamer Hasbrouck	Pier 35, North River, New York	3		do	do	Do.
	9	Steamer Redfield	do			do	do	Do.
	9	Steamer Ansonia	do	5		do	do	Do.
	9	Barge Chas. Spear	do	1		do	do	Do.
	9	Loeb Brothers	47th street between 1st avenue and East River, N. Y.	12		do	Fair	Do.
	10	Isaac Frank	49th street and East River, New York	6		do	Good	Do.
	10	J. Hetch	172 Delancy street, New York	9		do	do	Do.
	10	Sleck	do	7		do	do	Do.
	11	John Gillagar	87th street and 5th avenue, New York	14		do	do	Do.
	11	Mrs. O'Brien	103d street and 5th avenue, New York	10		do	do	Do.
	12	Mrs. Cannon	98th street and 4th avenue, New York	3		do	do	Do.
	12	Mrs. Regan	95th street and 4th avenue, New York	10		do	do	Do.
	12	Mrs. Shea	94th street and Madison avenue, New York	2		do	do	Do.
	12	John Cunningham	do	6		do	do	Do.
	13	Pat. Collins	91st street and Madison avenue, New York	14		do	do	Do.
	13	M. Duffy	96th street and 4th avenue, New York	6		do	do	Do.
	13	E. Kearney	96th street and Lexington avenue, New York	6		do	do	Do.
	13	Taylor	Lexington av. betw. 95th and 96th streets, New York	2		do	do	Do.
	9	Marks	47th street near 1st avenue, New York	6		do	do	Do.
	9	Steamer Catskill	Pier 34, North River, New York	52		do	do	Do.
	15	L. Coyne	1425 Avenue A	1		do	do	Do.
	15	Julia Buckley	73d street and East River, New York	3		do	do	Do.
	15	Mrs. McGee	71st street and Avenue A	1		do	do	Do.
	16	Steamer Catskill	Pier 34, North River, New York	31		do	do	Do.
	16	Steamer Ansonia	do	13		do	do	Do.
	16	Steamer Redfield	Pier 35, North River, New York	8		do	do	Do.
	16	Barge Chas. Spear	do	6		do	do	Do.
	19	S. Kolowsky	Tunnel avenue, New York	14	11	Fair	Fair	Do.
	20	Geo. Ringler	92d street between 2d and 3d avenues, New York	3		Good	Good	Do.
	20	Stahl	120th street and Pleasant avenue, New York	11		do	do	Do.
	20	John Distela	103d street and 2d avenue, New York	8		do	do	Do.
	2	A. Crook	37th street w. of 11th avenue, New York	12		Fair	do	C. B. Michener.

	2	Hugh Drennon	210th street and Kingsbridge road, New York	30		Good	do	Do.
	3	A. Siegworth	538 W. 40th street, New York	7		do	do	Do.
	3	J. Corbett	207th street and Kingsbridge road, New York	32		do	do	Do.
	4	M. Clancy	165 W. 10th avenue, New York	10		do	do	Do.
	4	John Credan	158th street e. Saint Nicholas avenue, New York	8		do	do	Do.
	6	Chas. S. Sutherland	Monroe avenue, New York	21		Very good	Very good	Do.
	9	A. Odenheimer	S. 40th street, w. 11th avenue, New York	3		Good	Good	Do.
	9	M. Duffy	133d street near Saint Nicholas avenue, New York	6		Very good	Very good	Do.
	9	Michael McGun	S. 37th street w. 11th avenue, New York	1		Good	Good	Do.
	10	Skelly & Fogarty	14th street w. 9th avenue, New York	5		do	do	Do.
	10	M. Tobin	31st street and 11th avenue, New York	1		do	Dying	Do.
	12, 13	James Stillman	Sing Sing	46		Very good	Very good	Do.
Dec.	15	S. McClintock	Between 112th and 113th streets, New avenue, N. Y.	12		do	do	Do.
	15	Mrs. English	37th street, near North River, New York	8		do	do	Do.
	17	A. Crooks	S. 37th street, near 11th avenue, New York	12		Poor	Fair	Do.
	17	M. Bruner	110th street and Boulevard, New York	12		Good	Good	Do.
	1	Union Stock Yards	60th street and North River, New York					Do.
	5	do	do					Do.
	8	do	do				Good	Do.
	1	do	do				do	Do.
	16	do	do			Good	Fair	Do.
	20	do	do			do	Good	Do.
	23	John Long	408 E. 63d street, New York	3		do	do	Do.
	22	Henry Nauman	321 E. 60th street, New York	1		do	do	Do.
	23	Steamer Hasbrouck	Pier 35, North River, New York	6			do	Do.
	23	John Rheinfrank	Corner 3d and Goerck streets, New York	2		Good	do	Do.
	24	J. Hetch	173 Delancey street, New York	8		do	do	Do.
	24	J. Slick	do	8		do	do	Do.
	26	P. Collins	2d avenue, between 91st and 92d streets, New York	6		do	do	Do.
	26	Corbett	421 E. 120th street, New York	3		do	do	Do.
	27	Burns	98th street and East River, New York	11		do	do	Do.
	27	P. King	273 E. 98th street, New York	22		Fair	Fair	Do.

INSPECTIONS ON LONG ISLAND, NEW YORK.

June	16	J. B. Wharry	906 Putnam avenue, Brooklyn	34				W. B. E. Miller.
	17	Henry Jarvis	Woodbine street, Brooklyn	30	2			Do.
	18	Jacob Walker	1047 Flushing avenue, Brooklyn	8	3			Do.
	18	James Baslet	do	27	3	Very bad	Very bad	Do.
	18	Mr. Dahl	Johnson avenue, near Flushing, Brooklyn	33	3	Very filthy		Do.
	18	Fredricka Schim	Flushing avenue, near Flushing, Brooklyn	32	3	Very bad		Do.
	19	Mike Green	Meserole street, Brooklyn	18	2			Do.
	19	Bridget Balfe	Evergreen avenue, corner Warfield street, Brooklyn	8	2	Very filthy		Do.
	19	Hugh Kenney	39 Jacob street, Brooklyn	38			All well	Do.
	20	George Pender	172 Woodbine street, Brooklyn	29		Very clean	do	Do.
	20	Kate Savage	99 Ralph avenue, Brooklyn	13	3			Do.
	21	J. Dobbins	Penny Bridge, Brooklyn	28	6	Very bad		Do.
	23	Nicholas Reamer	39th street and 8th avenue, Brooklyn	10			Healthy	Do.

Investigation of contagious pleuro-pneumonia—Inspections on Long Island, New York—Continued.

Date.		Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.								
June	23	John McGuire	38th street and 8th avenue, Brooklyn	18			Healthy	W. B. E. Miller.
	23	J. P. Errickson	39th street and 8th avenue, Brooklyn	4			do	Do.
	23	Eugene Sullivan	420 Columbia street, Brooklyn	1	1			Do.
	24	Ferdinand Roth	Windsor terrace, Flatbush	42	6			Do.
	25	Mrs. Callihan	31 Sullivan street, Brooklyn	2		Filthy	Healthy	Do.
	25	Philipp Pflawn	57th street and 6th avenue, Brooklyn	21			do	Do.
	26	Paddy McGowan	529 Bergen street, Brooklyn	5			do	Do.
	26	John Dalton	Henry and Huntington streets, Brooklyn	2		Filthy	do	Do.
	26	James Flynn	38th street and 8th avenue, Brooklyn	16	(?)			Do.
	26	Henry Scherff	42d street and 8th avenue, Brooklyn	8	2			Do.
	27	Mr. Chitwin	138 Cooper street, Brooklyn	18	1			Do.
	27	Mr. Gouse	Central street and Woodbine avenue, Brooklyn	23	1			Do.
	28	Henry Weidmaier	Center and Cooper streets, Brooklyn	3	1			Do.
	30	Anon Berethwisch	42d street and Bay Ridge, Brooklyn	21			Healthy	Do.
	30	James Murphy	39 40th street, Brooklyn	4			do	Do.
July	1	Mike Maloney	52 4th street, Brooklyn	7		Clean	do	Do.
	1	Martin Walsh	54 4th street, Brooklyn	2		do	do	Do.
	1	Mary Farrell	66 4th street, Brooklyn	3	1	Filthy	Fair	Do.
	1	Ellen Morrissey	96 4th street Brooklyn	2		do	do	Do.
	1	Thomas Birady	24 4th street, Brooklyn	1		Fair	do	Do.
	1	James Henry	18 4th street, Brooklyn	1		Very clean	Very good	Do.
	1	Elizabeth Lahey	70 4th street, Brooklyn	1		Good	Healthy	Do.
	2	Peter Blake	220 N. 9th street, Brooklyn	1		do	do	Do.
	2	Patrick Burns	61 N. 8th street, Brooklyn	2		do	do	Do.
	2	Philipp Duff	4th street, near N. 10th, Brooklyn	5		do	do	Do.
	2	Robt. Dixon	Cor. 1st and N. 8th streets, Brooklyn	1		do	do	Do.
	2	Patrick Flood	470 1st street, Brooklyn	3		do	do	Do.
	2	Andrew McDermott	223 N. 8th street, Brooklyn	1		do	do	Do.
	2	Hugh Monaghan	95 N. 9th street, Brooklyn	1		do	do	Do.
	2	John Collins	332 3d street, Brooklyn	3		do	do	Do.
	2	Peter Berg Hansen	78 N. 6th street, Brooklyn	6		do	do	Do.
	2	Richard Stark	N. 12th street, Brooklyn	16		do	do	Do.
	2	Wm. Hayes	N. 12th street, near 5th, Brooklyn	9		do	do	Do.
	3	Mary Campbell	288 Union avenue, Brooklyn	3		do	do	Do.
	3	John Doyle	311 Union avenue, Brooklyn	1		do	do	Do.
	3	James Flanaghan	415 Leonard street, Brooklyn	5		do	do	Do.
	3	Margaret Mayer	365 Lorimer street, Brooklyn	2		do	do	Do.
	3	Solomon May	212 Manigar street, Brooklyn	3		do	do	Do.

3	Michael McGerny	282 Leonard street, Brooklyn	1		do	do	Do.
3	H. Linde	345 Lorimer street, Brooklyn	1		do	do	Do.
3	Mike Thornton	19 Richardson street, Brooklyn	5		do	do	Do.
3	John Dippell	287 Meserole street, Brooklyn	18	1	do	do	Do.
4	Michael Mentheimer	155 Seigel street, Brooklyn	13		do	do	Do.
4	Solomon Miller	156 Seigel street, Brooklyn	2		do	do	Do.
4	Joseph Ladoux	126 Seigel street, Brooklyn	10		do	do	Do.
4	John Leitz	124 Seigel street, Brooklyn	1		do	do	Do.
4	Lazarus Neil	48 Seigel street, Brooklyn	2		do	do	Do.
4	Louis Bithing Meyer	270 Ten Eyck street, Brooklyn	7		do	do	Do.
4	Simon Moog	164 Scholes street, Brooklyn	2		do	do	Do.
4	F. Obrigin	19 Garden street, Brooklyn	3		do	do	Do.
5	Magdalen Guyer	56 Moore street, Brooklyn	2		do	do	Do.
5	John Kitts	100 Moore street, Brooklyn	2		do	do	Do.
5	Max Kirchmeyer	34 Moore street, Brooklyn	4		do	do	Do.
5	August Roder	30 Moore street, Brooklyn	2		do	do	Do.
5	Jacob Strau	20 Moore street, Brooklyn	5		do	do	Do.
5	Mrs. Gans	Central and Woodbine streets, Brooklyn	23	1	do	do	Do.
5	R. Horker	Casper and Central avenue, Brooklyn	1		do	do	Do.
5	Jacob Schmidt	275 Mayer street, Brooklyn	4		do	do	Do.
5	Adam Schneider	309 Mayer street, Brooklyn	3		do	do	Do.
5	Philip Wackerman	348 Maniger street, Brooklyn	6		do	do	Do.
5	Henry Darley	7 Walton street, Brooklyn	1		do	do	Do.
7	Caspar Bock	264 Johnson avenue, Brooklyn	2		Filthy	Bad	Do.
7	Philip Schneider	268 Johnson avenue, Brooklyn	2		Fair	Fair	Do.
7	Valentine Bauer	287 Wallabout street, Brooklyn	5		Good	Good	Do.
7	Margaret Burns	292 Wallabout street, Brooklyn	8		Filthy	Fair	Do.
7	Catharine Commer	174 Wallabout street, Brooklyn	1		do	Bad	Do.
7	Francis Frulick	287 Wallabout street, Brooklyn	5		Very filthy	do	Do.
7	P. McKenney	211 Wallabout street, Brooklyn	1		Good	Good	Do.
7	M. McMann	184 Wallabout street, Brooklyn	1		do	do	Do.
7	Henry Meyer	307 Wallabout street, Brooklyn	1		Filthy	Fair	Do.
7	Kate Rosenmayer	305 Wallabout street, Brooklyn	8		Good	Good	Do.
7	Margaret Mitchell	288 Wallabout street, Brooklyn	8		do	do	Do.
7	Henry Newman	328 Rutledge street, Brooklyn	2		do	do	Do.
7	Samuel Ritchie	331 Rutledge street, Brooklyn	7		do	do	Do.
8	F. L. Butzgy	38 Wyckoff avenue, Brooklyn	24	2	do	do	Do.
8	Edmund Stone	Wyckoff ave. and Trautman st., Brooklyn 2	5	2	Filthy	do	Do.
8	Joseph Rupert	Myrtle street, Brooklyn	8		Fair	do	Do.
8	Frank Rupert	do	5	2	do	do	Do.
8	Herman Gendes	1231 Flushing avenue, Brooklyn	1		do	do	Do.
8	C. E. Vansise	Flushing ave., near Johnson ave., Brooklyn	42		do	do	Do.
8	Peter Wyckoff	do	2		do	do	Do.
8	Amill Murkett	50 Prospect street	29		do	do	Do.
8	John Dilman	827 Evergreen avenue	3	1	do	do	Do.
8	Abraham Harpbst	316 Rutledge street	7		do	do	Do.
8	George A. Pindar	160 Ivy street	27	7	do	do	Do.
8	Edmund Jarvis	Ivy street	26	2	do	do	Do.
8	John Carey	Wyckoff avenue	45	2	do	Very good	Do.
8	John Weiman	do	8		do	do	Do.
8	John Gorr	418 Green street, Ridgewood	11		do	do	Do.
8	Charles Gardiner	North street	9		do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections on Long Island, New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884. July 8	Wm. Kronenberger	Irving avenue and Stockholm street	6	1	Fair	Very good	W. B. E. Miller.
8	Peter Kelley	do	9		do	do	Do.
8	G. H. Hubbs	Newton, near Flushing avenue	106		Excellent	Excellent	Do.
8	Edward Hubbs	do	52		Good	Good	Do.
8	Catharine Quinn	Fifth avenue and St. John's Place	1		Filthy	Healthy	Do.
8	D. McGuinness	do	10			do	Do.
8	Mr. Berg	94 Fifth avenue	1			do	Do.
8	Miles Murphy	24 Garfield Place	4			do	Do.
8	Paddy McCurran	1st, near 4th avenue	1			do	Do.
8	M. Gorrigan	198 20th street, Brooklyn, N. Y	2			Healthy	Do.
8	Mary Kelley	210 20th street, Brooklyn, N. Y	2			do	Do.
8	M. Schaeffer	188 20th street, Brooklyn, N. Y	1			do	Do.
8	Mary Clear	156 20th street, Brooklyn, N. Y	8		Good	Good	Do.
8	Ernest Rubus	148 20th street, Brooklyn, N. Y	8	2	do	do	Do.
8	Patrick Joyce	47 20th street, Brooklyn, N. Y	4		do	do	Do.
8	Bridget Kennedy	202 21st street, Brooklyn, N. Y	8		do	do	Do.
8	Mrs. Riley	42d street near 1st avenue, Brooklyn, N. Y	1		do	do	Do.
8	J. P. Dickinson	49th street, near 1st avenue, Brooklyn, N. Y	1		do	do	Do.
8	Mr. Williams	do	1		do	do	Do.
8	Humphry McCarty	41st street and 3d avenue, Brooklyn, N. Y	7		do	do	Do.
8	Jas. Hafferman	106 38th street, Brooklyn, N. Y	18		Filthy	Bad	Do.
8	Mrs. Garrigan	Near 4th avenue, Brooklyn, N. Y	2		Good	Good	Do.
8	Mr. Gilmartin	20th street, near 4th avenue, Brooklyn, N. Y	1		do	do	Do.
8	James L. Martin	59th street and Gowanus Bay, Brooklyn, N. Y	7	1	do	do	Do.
8	Mr. Minss	do	8		do	do	Do.
8	Several owners	On lots, 59th street and 3d avenue, Brooklyn, N. Y	26		do	do	Do.
8	Tyson Bond	Foot of 53d street, Brooklyn, N. Y	20	1	Very good	Very good	Do.
8	Mr. Williams	Foot af 49th street, Brooklyn, N. Y	4		do	do	Do.
8	James Edwards	3d avenue and 49th street, Brooklyn, N. Y	1		do	do	Do.
8	John Dipple	287 Meserole street, Brooklyn, N. Y	22	2	do	do	Do.
8	Mrs. J. Joice	180 Hamburg avenue, Brooklyn, N. Y	15	1	do	do	Do.
8	Joseph Rumple	Cor. Wyckoff ave. and Jefferson st., Brooklyn, N. Y	4		do	do	Do.
19	James Carroll	do	13	2	do	do	Do.
28	Mr. Zabriskie	Church lane, Flatbush, N. Y	2		do	do	Rowland & Hawk.
28	Mr. Shultz	Near Flatbush avenue, Flatbush, N. Y			do	do	Do.
29	John Morrow	East Broadway, Flatbush, N. Y	14		Bad	do	Do.
29	J. Mullenuex	do	17		Fair	do	Do.
29	H. Muller	East Broadway, Flatbush, N. Y	1		Very good	Good	Do.
29	T. Fitzpatrick	Grand street, Flatbush, N. Y	4		do	do	Do.

29	Jessie Hallahan	Erasmus street, Flatbush, N. Y	6		Bad	do	Do.
29	Thomas McKenney	Prospect street, Flatbush, N. Y	11		Good	do	Do.
30	John Moran	Upper end of Martin's lane, N. Y	4		do	do	Do.
30	D. Kellner	Chester street and 12th avenue, Flatbush, N. Y	35		Fair	Fair	Do.
30	Fredk. Behrens	Franklin avenue, Flatbush, N. Y	13		Good	Good	Do.
30	Lewis Britz	Caton avenue, Flatbush, N. Y	8		Very good	do	Do.
30	E. R. Smith	Caton Park, Flatbush, N. Y	1		do	do	Do.
30	William Thoms	Caton, Flatbush, N. Y	2		do	do	Do.
30	J. Hayslips	Church lane and Boulevard, Flatbush, N. Y	85	1	Bad	do	Do.
30	Thomas McGirney	Church lane Flatbush, N. Y	8		Good	do	Do.
30	Geo. Cook	Church lane, Flatbush, N. Y	9		do	do	Do.
30	Christian Hoffman	Martin's lane, Flatbush, N. Y	16		do	do	Do.
30	John Balan	do	1		do	do	Do.
31	Jas. Busby	Newkirk avenue, Flatbush, N. Y	4		do	do	Do.
31	Patrick Oats	do	8		Very good	Very good	Do.
31	J. W. Wilder	Near Newkirk avenue, Flatbush, N. Y	11		do	do	Do.
31	Mr. Elger	Old Coney Island road, Flatbush, N. Y	1		do	do	Do.
31	Mr. Hogan	do	2		do	do	Do.
31	A. Uhlman	do	2		do	do	Do.
Aug. 1	A. Fish	Near Greenwood, Flatbush, N. Y	3		do	do	Do.
1	M. Purse	Near Greenwood avenue, Flatbush, N. Y	1		do	do	Do.
1	Fredk. Carles	do	2		do	do	Do.
1	Ferdinen Roth	E. 5th street, Flatbush, N. Y	39	9	do	do	Do.
1	Mr. Henderson	Seeley street, Flatbush, N. Y	1		do	do	Do.
1	Iven Cumiskey	Adams street, Flatbush, N. Y	5		do	do	Do.
1	W. E. Murphy	Greenwood, Flatbush, N. Y	1		do	do	Do.
1	Mr. Smith	Vanderbilt avenue, Flatbush, N. Y	1			Good	Do.
1	Mr. James Hagerty	Flatbush avenue, Flatbush, N. Y	4		Good	do	Do.
1	John B. Halding	do	1		do	do	Do.
1	Patrick Fitzpatrick	do	8		do	do	Do.
1	Mr. Jones	do	1		Bad	do	Do.
1	John Vandevere	do	4		Good	do	Do.
4	Wm. D. Geddis	Malbone street, Flatbush, N. Y	12		do	do	Do.
4	E. Bennet	Montgomery street, Flatbush, N. Y	30	3	do	Fair	Do.
4	Ottis Velia	Cedar street, Flatbush, N. Y	1		do	Good	Do.
4	Thomas Cassidy	Franklin avenue, Flatbush, N. Y	1		do	do	Do.
4	Mr. Colman	E. New York avenue, Flatbush, N. Y	1		do	do	Do.
4	John Clancy	do	36		Filthy	Bad	Do.
4	John Brady	do	17		Good	Good	Do.
4	Mr. Hussey	Near E. New York avenue, Flatbush, N. Y	1		do	do	Do.
4	John Leopard	do	2		do	do	Do.
4	Wm. Crozier	William street, Flatbush, N. Y	22	(?)	(?)	(?)	Do.
4	Mrs. Ellen Divine	do	2		Bad	Good	Do.
4	John Hoffman	do	4		do	do	Do.
4	Wm. Shaferman	Troy avenue, Flatbush, N. Y	1		do	do	Do.
4	J. Sheredon	do	2		do	do	Do.
4	Mr. Larkinor	Cullin street, Flatbush, N. Y	1		do	do	Do.
4	John Hall	do	1		do	do	Do.
4	John Rider	Albany avenue, Flatbush, N. Y	1		do	do	Do.
4	Andrew Darhay	do	2		do	do	Do.
4	Henry Tyson	Hunterfly road, Flatbush, N. Y	4		Good	do	Do.
6	P. Murphy	Rochester avenue, Flatbush, N. Y	20		do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections on Long Island, New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.							
Aug. 6	R. Kelloy	President street, Flatbush, N. Y	30	1	Good	Good	Rowland & Hawk.
6	Thomas Warren	Union street, Flatbush, N. Y	9	1	do	do	Do.
6	S. S. Vandevere	E. New York avenue, Flatbush, N. Y	1			do	Do.
6	J. W. Mehl	Hunterfly road, Flatbush, N. Y	29	1	Good	do	Do.
6	John Kizer	do	2	1		Bad	Do.
6	Mrs. Walsh	Carl street, Flatbush, N. Y	4		Good	Good	Do.
6	John Fox	Utica avenue, Flatbush, N. Y	21			do	Do.
6	Mr. Lines	Troy street, Flatbush, N. Y	6			do	Do.
7	John Hallan	Cedar street, Flatbush, N. Y	4			do	Do.
7	James Kelly	do	3		Good	do	Do.
7	John Velia	do	1		do	do	Do.
7	Thomas Trimmer	Franklin avenue, Flatbush, N. Y	7		do	do	Do.
7	Edward Sermot	E. New York avenue, Flatbush, N. Y	61		Filthy	do	Do.
8	A. Stuart	President street, Flatbush, N. Y	6		Bad	do	Do.
8	Mrs. M. Culbert	do	5		Good	do	Do.
8	John Force	Carl street, Flatbush, N. Y	10		do	do	Do.
8	Mr. Morris	do	1		do	do	Do.
8	Henry Mohahan	Clarkson street, Flatbush, N. Y	6		Filthy	do	Do.
8	William Williamson	E. Broadway, Flatbush, N. Y			Good	do	Do.
8	P. Nephus	do	8		do	do	Do.
8	Nicholas Cummings	do	1		do	do	Do.
8	T. Pherous	do	1		do	do	Do.
8	John Sudam	Kenarcy street, Flatbush, N. Y	2		do	do	Do.
8	George Van Wyck	do	2		do	do	Do.
8	R. Rempson	Hunterfly road, Flatbush, N. Y	1		do	do	Do.
8	P. Rempson	do	2		do	do	Do.
8	S. Lots	do	2		do	do	Do.
8	J. B. Cowenhoven	Flatlands Neck road, Flatbush, N. Y	8		do	do	Do.
8	D. Cowenhoven	do	1		do	do	Do.
8	W. G. K. Cowenhoven	do	2		do	do	Do.
8	H. Rump	Flatlands Neck road, Flatbush, N. Y	2		do	do	Do.
8	C. Ditmus	do	1		do	do	Do.
8	Henry Cameron	do			do	do	Do.
8	Jacob Henry	Camerscy lane, Flatbush, N. Y	8			do	Do.
8	Patrick Curley	Prospect street, Flatbush, N. Y	2			do	Do,
8	M. Mills	do	1			do	Do.
8	Thomas Lanahan	Johnson's Place, Flatbush, N. Y	4			do	Do.
8	Mrs. M. Carrell	Prospect street, Flatbush, N. Y	2			do	Do.

	8	Mrs. P. O'Connelly	Butler street, Flatbush, N. Y	7			do	Do.
	8	M. Lots	Flatbush avenue, Flatbush, N. Y	2			do	Do.
	8	J. L. Rider	Flatlands Neck road, Flatbush, N. Y	2		Good	do	Do.
July	26	Sundry owners	12th street and 2d avenue, Brooklyn, N. Y	43			do	William B. E. Miller.
	26	do	12th street and 3d avenue, Brooklyn, N. Y	81			do	Do.
	26	Robert Ferris	172 12th street	4	1	Fair	Fair	Do.
	26	Patrick Gregory	12th street, near 2d avenue	3	1	Filthy	Bad	Do.
	28	James Martin	100 West 8th street	4	1	do	Fair	Do.
	28	Kate Martin	9th street and 2d avenue	6	8	do	Bad	Do.
	28	John Delmar	8th street and 2d avenue	14	2	do	do	Do.
	28	Michael Martin	do	18	5	Good	Good	Do.
	30	Sundry owners	10th street and 2d avenue	24	1		do	Do.
	30	do	10th street and 3d avenue	19				Do.
	30	Mrs. Grant	Foot of 10th street	11		Fair	Good	Do.
	31	James Strausse	Hotel, Central avenue	1	1			Do.
	31	Adam Rothair	Central avenue, near Evergreen Cemetery	13		Good	Good	Do.
	31	John Chatwin	do	18		do	do	Do.
	31	Sundry owners	do	57			do	Do.
Aug.	1	Samuel Hart	577 Bergen street	8		Good	do	Do.
	1	W. Davis	935 Bergen street	24	6	Poor	Fair	Do.
	1	Fred. Kuckak	1984 Bergen street, Brooklyn, N. Y	24	4	Fair	Bad	Do.
	2	Michael Kernan	1691 Fulton street, Brooklyn, N. Y	22	2	Good	Good	Do.
	2	A. Knapp	1153 Saint Mark's street, Brooklyn, N. Y	17	8	Fair	do	Do.
	2	Richard Bennett	do	82		Good	do	Do.
	2	Sundry owners	On lots, Saint Mark's street, Brooklyn, N. Y	86		do	do	Do.
	5	George A. Pindar	160 Ivy street, Brooklyn, N. Y	27	6	do	do	Do.
	11	James Gern	Cor. Cooper ave. and Lafayette st., Brooklyn, N. Y	4		do	do	Do.
	11	Pat. McBride	Cypress Hills road, Brooklyn, N. Y	14		Fair	Fair	Do.
	11	George Swartz	do	23		Good	Good	Do.
	11	Sundry owners	do	5		Fair	Fair	Do.
	12	do	On lots, Brooklyn, N. Y	54	(?)		Bad	Do.
	12	James Gascoine	Fresh Pond road and Myrtle street, Brooklyn, N. Y	95		Good	Good	Do.
	12	W. L. Smith	Fresh Pond road, Brooklyn, N. Y	19		do	do	Do.
	12	S. Jarvis	Cooper avenue, Brooklyn, N. Y	17		do	do	Do.
	12	A. J. Layton	Cor. Cooper and Cypress avenues, Brooklyn, N. Y	84		do	do	Do.
	13	Sundry owners	Glendale road, Brooklyn, N. Y	51				Do.
	13	T. P. Ryan	do	26		Good	Good	Do.
	13	J. B. McGann	Myrtle avenue and Glendale, Brooklyn, N. Y	23		do	do	Do.
	13	H. Ring	do	6		do	do	Do.
	14	G. A. Vansise	Glendale road, near Brooklyn, N. Y	83		do	do	Do.
	14	Geo. A. Bennett	Cor. Stone and Liberty avenues, Brooklyn, N. Y	24		do	do	Do.
	14	Mike Conners	1455 Troy avenue, Brooklyn, N. Y	6		do	do	Do.
	14	Mrs. Cahill	On lot, Albany avenue, Brooklyn, N. Y	2		do	do	Do.
	14	Sundry owners	On lot, near Albany avenue, Brooklyn, N. Y	86			do	Do.
	15	Nathaniel Smith	Liberty avenue, East New York	14	1			Do.
	15	Jesse Matt	Christopher st., near Liberty ave., East New York	80	1	Good	Good	Do.
	15	A. T. Badell	Sackman st. and Liberty ave., East New York	29		do	do	Do.
	15	Alfred Wanser	do	9		do	do	Do.
	15	John Buffett	Sackman street and Liberty avenue, East New York	41		do	do	Do.
	15	Charles Ryan	Sackman street, corner Baltic ave., East New York	45	1	do	do	Do.
	16	Jesse W. Hewlett	Sackman street and Liberty avenue, East New York	26	1	do	do	Do.
	16	Martin Vansise	Sackman street, East New York	81		do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections on Long Island, New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stables.	Condition of animals.	Name of inspector.
1884. Aug. 16	Carman Combs	Cooper avenue, East New York	42		Good	Good	William B. E. Miller.
16	Sundry owners	On lots, Atlantic and Fulton avennes, Brooklyn, N. Y	66				Do.
16	do	Ridgwood road and Cooper avenue, Brooklyn, N. Y	3			Good	Do.
11	Jeffrey Van Wyck	King's Highway, Flatlands, N. Y	2		Good	do	Rowland & Hawk.
11	W. Bush	Flatlands avenue, Flatlands, Brooklyn	1			do	Do.
11	John A. Bush	do	1			do	Do.
11	T. Burgen	Flatlands road, Flatlands, N. Y	1			do	Do.
11	J. L. Rider	do	2		Good	do	Do.
11	J. Rider	do	4		do	do	Do.
11	M. Murray	do	1			do	Do.
11	Garnet Burgen	do	1			do	Do.
11	P. Walkman	Ocean avenue, Flatlands, N. Y	2		Good	do	Do.
11	Mrs. Bennet	do	1		do	do	Do.
11	James Mack	do	1			do	Do.
11	Wm. Schey	do	1		Good	do	Do.
11	J. H. Fitzpatrick	do	1		do	do	Do.
11	J. Lang	do	1			do	Do.
11	P. Collins	Ocean avenue, Flatbush, N. Y	1			do	Do.
11	John Collins	do	2			do	Do.
11	M. Olman	Old Coney Island road, Flatbush, N. Y	1			do	Do.
11	N. Shirt	do	1			do	Do.
11	John Weedner	do	2		Good	do	Do.
11	John Gunas	do	1		Filthy	do	Do.
11	G. Kline	Old Coney Island road, Gravesend, N. Y	2			do	Do.
11	John Dempsey	Old Coney Island road, New Utrecht, N. Y	6		Good	do	Do.
11	John Whitners	do	2			do	Do.
11	Mrs. M. Johnson	do	8			do	Do.
11	Matthew Gerrin	do	1		Good	do	Do.
11	John Luydam	do	2			do	Do.
11	Mr. Dunley	Foster avenue, New Utrecht, N. Y	1			do	Do.
11	Chas. Sephas	Lawrence avenue, New Utrecht, N. Y	1		Good	do	Do.
11	James Busby	Webster avenue, New Utrecht, N. Y	1			do	Do.
11	Wm Slate	Washington avenue, New Utrecht, N. Y	1			do	Do.
11	G. Fay	do	1		Good	do	Do.
11	P. Gu en	Foster avenue, New Utrecht, N. Y	2			do	Do.
11	L. Boilan	Franklin avenue, New Utrecht, N. Y	2			do	Do.
11	John Shea	Washington avenue, New Utrecht, N. Y	2			do	Do.
11	R. Perrin	Franklin avenue, New Utrecht, N. Y	1			do	Do.
11	P. Ke nan	do	1		Good	do	Do.

11	J. Keenan	do	2		do	do	Do.
11	James Lary	Webster avenue, New Utrecht, N. Y	8		Bad	do	Do.
11	T. Rutherford	do	2			do	Do.
11	H. Wiggins	Washington avenue, New Utrecht, N. Y	2			do	Do.
11	Patrick Walts	do	1			do	Do.
12	J. Satts	Flatbush avenue, Flatbush, N. Y	2		Good	do	Do.
12	V. B. McGan	Ocean avenue, Flatbush, N. Y	2		do	do	Do.
12	M. Limney	Coney Island road, Flatlands, N. Y				do	Do.
12	T. Gillfathers	Near Coney Island road, Flatlands, N. Y	1			do	Do.
12	J. Van Western	Flatbush avenue, Flatlands, N. Y	1			do	Do.
12	W. H. Allen	do	1		Good	do	Do.
12	J. Van Wick	Near Coney Island road, Flatlands, N. Y	2		do	do	Do.
12	A. Ditmus	do	2			do	Do.
12	A. H. Van Brunt	do	4		Bad	do	Do.
12	F. Sheppard	do	1		do	do	Do.
12	E. A. Hubbard	Near Flatbush avenue, Flatlands, N. Y	1		do	do	Do.
12	J. P. Prout	Mill lane, Flatlands, N. Y	21	1	Good	do	Do.
12	W. I. Wycoff	Near Satts lane, Flatlands, N. Y	1		do	do	Do.
12	Weil & Weedman	Near Flatbush avenue, Flatlands, N. Y	2		do	do	Do.
12	N. P. McLacklin	Near Satts lane, Flatlands, N. Y	2		do	do	Do.
12	W. Brown	Flatbush avenue, Flatbush, N. Y	1		do	do	Do.
12	A. A. Brown	Corner Franklin avenue, Flatbush, N. Y	1		do	do	Do.
12	M. Westforce	Near Flatbush avenue, Flatbush, N. Y	1		do	do	Do.
12	G. Stryker	do	1		do	do	Do.
12	J. Myatti	do	1		do	do	Do.
12	J. Zelat	Ocean avenue, Flatbush, N. Y	2		do	do	Do.
12	A. Satts	Flatbush avenue, Flatbush, N. Y	1		do	Fair	Do.
12	Mrs. F. Durea	do	2		do	Good	Do.
12	P. Ford	Satts lane, Flatlands, N. Y	4		do	do	Do.
12	D. H. Satts	do	1		do	do	Do.
12	J. Van Dyke	do	1		do	do	Do.
12	C. H. Dwyer	do	1		do	do	Do.
12	J. G. McGan	Ocean avenue, Flatbush, N. Y	1		do	do	Do.
12	Mr. Ditmus	Near Butler street, Flatbush, N. Y	2		do	do	Do.
12	C. Callahan	do	1		do	do	Do.
12	C. Shaffer	Grand street, Flatbush, N. Y	2		do	do	Do.
12	M. Connis	do	1		do	do	Do.
12	Mr. Stapleton	Flatbush avenue, Flatbush, N. Y	1		do	do	Do.
12	R. G. Strong	East Broadway, Flatbush, N. Y	1		do	do	Do.
13	M. Dwyer	Canarsie street, Flatlands, N. Y	1		do	do	Do.
13	A. C. Urah	do	2		do	do	Do.
13	G. Konenhoven	do	8		do	do	Do.
13	M. Konenhoven	do	2		do	do	Do.
13	D. Whalen	do	1		do	do	Do.
13	H. Sudam	do	1		do	do	Do.
13	J. Skelly	do	1		do	do	Do.
13	L. H. Smith	do	1		do	do	Do.
13	J. Flancy	do	2		do	do	Do.
13	J. Sudam	Near Canarsie street, Flatlands, N. Y	1		do	do	Do.
13	J. Skiotman	Near Washington avenue, Gravesend, N. Y	2		do	do	Do.
13	M. Gates	do	1		do	do	Do.
13	W. Slate	Washington avenue, Gravesend, N. Y	2		do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections on Long Island, New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.							
Aug. 13	John Shea	Washington avenue. Gravesend, N. Y	3		Good	Good	Rowland & Hawk.
13	P. Guerin	Near Foster avenue, Gravesend, N. Y	1		do	do	Do.
13	M. Feeney	Foster avenue, Gravesend, N. Y	1		do	do	Do.
13	G. Clark	do	2		do	do	Do.
13	H. McCabe	do	1	1	do	do	Do.
13	P. H. Ward	Near Boulevard, Gravesend, N. Y	1		do	do	Do.
13	John Bergen	Boulevard, Gravesend, N. Y	1		do	do	Do.
13	W. McMann	do	2		do	do	Do.
13	Geo. Clark	do	2		do	do	Do.
13	A. Patterson	do	4		do	do	Do.
13	H. G. Freedman	do	1		do	do	Do.
13	W. Brown	do	2		do	do	Do.
13	W. Galleger	do	1		do	do	Do.
13	Mrs. E. Kelley	King's Highway, Flatlands, N. Y	3		do	do	Do.
13	John Kreyers	do	1		do	do	Do.
13	J. M. Sebellows	do	2		do	do	Do.
13	H. R. Wycoff	do	2		do	do	Do.
13	W. Bennett	do	1		do	do	Do.
13	B. J. Hitchers		1		do	do	Do.
13	J. McMonigans	Old Coney Island road, Gravesend, N. Y	1		do	do	Do.
13	M. Laurence	do	4		do	do	Do.
13	T. Furgison	do	3		do	do	Do.
14	Dr. Herd	Webster avenue, Parkville, N. Y	1		do	do	Do.
14	M. Martin	do	1		do	do	Do.
14	B. Bennett	Near Webster avenue, Parkville, N. Y	4		do	do	Do.
14	M. Sothern	do	2		do	do	Do.
14	T. Schenck	Flatbush road, New Utrecht, N. Y	3		do	do	Do.
14	John Cowenhoven	do	1		do	do	Do.
14	P. Cowenhoven	do	1		do	do	Do.
14	P. McNally	do	3		Filthy	do	Do.
14	T. Frice	do	1		Good	do	Do.
14	J. Cooper	do	1		do	do	Do.
14	T. McNally	do	3		do	do	Do.
14	W. Garland	do	2		do	do	Do.
14	R. J. Berry	do	3		do	do	Do.
14	P. Feeney	Franklin street, New Utrecht, N. Y	2		do	do	Do.
14	Henry Bennett	22d street, New Utrecht, N. Y	1		do	do	Do.
14	P. Libe	do	1		do	do	Do.

14	Thomas Flannigan	do	2		do	do	Do.
14	Otto Cobner	King's Highway, New Utrecht, N. Y	2		do	do	Do.
14	T. C. Van Pelt	do	1		do	do	Do.
14	J. Van Pelt, sr	Bath road, New Utrecht, N. Y	1		do	do	Do.
14	J. Van Pelt, jr	Near Bath road, New Utrecht, N. Y	1		do	do	Do.
14	Jeremiah Satts	Bath road, New Utrecht, N. Y	2		do	do	Do.
14	P. Hagerman	Near Bath road, New Utrecht, N. Y	1		do	do	Do.
14	J. Smydt	Bath road, New Utrecht, N. Y	1		do	do	Do.
14	Eford Sudam	do	1		do	do	Do.
14	T. Lauxman	do	1		do	do	Do.
14	Jerrie Satts	do	1		do	do	Do.
14	Dr. Roberts	do	19		do	do	Do.
14	A. Satts	do	1		do	do	Do.
14	R. Spedding	do	1		do	do	Do.
14	Mr. Sargent	do	2		do	do	Do.
14	G. Coweenhoven	do	2		do	do	Do.
14	J. Flalscraft	do	8		do	do	Do.
14	D. McMulligan	do	2		do	do	Do.
14	J. C. Satts	do	2		do	do	Do.
14	W. Cropsey	Breedy street, New Utrecht, N. Y	1		do	do	Do.
15	Unknown	Near 16th street, New Utrecht, N. Y	5		do	do	Do.
15	Mrs. McGuire	16th street, New Utrecht, N. Y	1		do	do	Do.
15	M. Donahue	do	1		do	do	Do.
15	J. Flinn	38th street, New Utrecht, N. Y	16		do	do	Do.
15	J. Miller	Near 39th street, New Utrecht, N. Y	11		do	do	Do.
15	H. C. Falscraft	Near plank road, New Utrecht, N. Y	2		do	do	Do.
15	M. Montfort	do	1		do	do	Do.
15	M. Wycoff	do	1		do	do	Do.
15	P. Mark	do	1		do	do	Do.
15	W. H. Crapsey	do	1		do	do	Do.
15	V. Voorhees	do	1		do	do	Do.
15	Joseph Kinceide	do	4		do	do	Do.
15	Fritz Clap	do	1		do	do	Do.
15	Frank Butler	do	1		do	do	Do.
15	A. Theil	do	1		do	do	Do.
15	M. Jodison	Near plank road, New Utrecht, N. Y	1		do	do	Do.
15	J. Degraw	do	1		do	do	Do.
15	J. Leubrv	do	2		do	do	Do.
15	J. E. Satts	do	2		do	do	Do.
15	Dr. De Mont	King's Highway, New Utrecht, N. Y	2		do	do	Do.
15	H. Smith	do	1		do	do	Do.
15	P. Walch	do	3		do	do	Do.
15	Geo. Van Brunt	do	2		do	do	Do.
15	J. F. Peluger	do	4		do	do	Do.
15	A. Goobner	do	3		do	do	Do.
15	M. Pingle	do	2		do	do	Do.
15	A. Bennett	do	8		do	do	Do.
15	N. Ferginson	Near King's Highway, New Utrecht, N. Y	3		do	do	Do.
15	A. Kinsler	Dennis lane, New Utrecht, N. Y	1		do	do	Do.
15	Geo. Elas	do	1		do	do	Do.
15	Unknown	Near Dennis lane, New Utrecht, N. Y	1		do	do	Do.
15	M. Mitchell	Bennett lane, New Utrecht, N. Y	1		do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections on Long Island, New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.							
Aug. 15	D. T. Renolds	Bennett lane, New Utrecht, N. Y	1		Good	Good	Rowland & Hawk.
15	J. Fullman	Stewart lane, New Utrecht, N. Y	2		do	do	Do.
15	P. Dennis	do	1		do	do	Do.
15	M. Shrader	Franklin lane, New Utrecht, N. Y	1		do	do	Do,
15	W. Shrader	do	1		do	do	Do.
15	J. Franks	do	1		do	do	Do.
15	Geo. Jardine	do	1		do	do	Do.
15	M. L. Paen	do	1		do	do	Do.
15	C. Osier	do	2		do	do	Do.
15	P. Smith	do	1		do	do	Do.
15	M. Reinmer	do	10		do	do	Do.
15	J. McGuire	Franklin avenue, New Utrecht, N. Y	18		do	do	Do.
15	I. McDougal	do	1		do	do	Do.
15	Patrick Coyle	Hamilton avenue, New Utrecht, N. Y	14		do	do	Do.
15	M. Zimmerman	17th street, New Utrecht, N. Y	1		do	do	Do.
15	J. Kimsington	505 Prospect avenue, New Utrecht, N. Y	2		do	do	Do.
18	Geo. Funk	Near 49th street, near Bay Ridge, N. Y	1		do	do	Do.
18	R. J. Wardler	Near 49th street, New Utrecht, N. Y,	1		do	do	Do.
18	H. Minson	do	1		do	do	Do.
18	Edwin Tylor	do	1		do	do	Do.
18	Geo. Winner	Near 58th street, New Utrecht, N. Y	3		do	do	Do.
18	M. Flider	do	1		do	do	Do.
18	P. Mack	66th street, New Utrecht, N. Y	1		do	do	Do.
18	M. J. Bergen	Near Hamilton avenue, New Utrecht, N. Y	5		do	do	Do.
18	Mr. Weinright	64th street, New Utrecht, N. Y	4		do	do	Do.
18	H. Kalman	66th street, New Utrecht, N. Y	5		do	do	Do.
18	James McKerry	65th street, New Utrecht, N. Y	11		do	do	Do.
18	James Louire	do	1		do	do	Do.
18	Geo. Hope	do	2		do	do	Do.
18	P. Lenhart	do	1		do	do	Do.
18	J. C. Kempton	Overton avenue, New Utrecht, N. Y	3		do	do	Do.
18	M. J. Wilses	Near Overton avenue, New Utrecht, N. Y	2		do	do	Do.
18	J. Taylor	Overton avenue, New Utrecht, N. Y	2		do	do	Do.
18	M. Hefferon	do	4		do	do	Do.
18	J. Wersan	do	1		do	do	Do.
18	D. Duscill	Main street, Bay Ridge, N. Y	3		do	do	Do.
18	Mr. Aspenwell	do	2		do	do	Do.
18	B. C. Townsend	3d avenue, Bay Ridge, N. Y	3		do	do	Do.
18	M. Thomas	do	2		do	do	Do.

19	W. Bennett	Near 3d avenue, Bay Ridge, N. Y	8		do	do	Do.
19	W. Perry	do	3		do	do	Do.
19	E. Kent	Near 2d avenue, Bay Ridge, N. Y	2		do	do	Do.
19	Alex. Champion	do	1		do	do	Do.
19	A. W. Bliss	do	3		do	do	Do.
19	M. E. Burnett	Bay Ridge, N. Y	3		do	do	Do.
19	A. McGlue	do	1		do	do	Do.
19	M. Bull	Near Bay Ridge, N. Y	1		do	do	Do.
19	J. McCay	do	2		do	do	Do.
19	M. Bennett	Shore road, Bay Ridge, N. Y	1		do	do	Do.
19	W. Bennett	do	3		do	do	Do.
19	D. Bennett	do	2		do	do	Do.
19	Rule & Brunt	do	4		do	do	Do.
19	C. White	do	2		do	do	Do.
19	James Mills	do	2		do	do	Do.
19	Mrs. Tumey	do	1		do	do	Do.
19	C. McNally	do	2		do	do	Do.
19	Mrs. Reupson	do	1		do	do	Do.
19	Mrs. Burns	do	3		do	do	Do.
19	P. Leager	do	4		do	do	Do.
19	J. Crowell	do	1		do	do	Do.
19	M. McLin	do	2		do	do	Do.
19	T. Parker	do	1		do	do	Do.
19	T. White	do	1		do	do	Do.
19	M. Granes	do	2		do	do	Do.
19	M Cunard	do	2		do	do	Do.
19	N. Farmer	do	2		do	do	Do.
19	Mrs. Detroit	do	2		do	do	Do.
19	M. Calman	Fort Hamilton avenue, Fort Hamilton, New York	1		do	do	Do.
19	Inebriates' Home	Near 92d street, Fort Hamilton, New York	3		do	do	Do.
19	M. Bullock	do	4		do	do	Do.
19	D. C. Bennett	do	7		do	do	Do.
19	John Furman	92d street, Fort Hamilton, New York	3		do	do	Do.
19	M. Kelly	Fort Hamilton avenue, Fort Hamilton, New York	1		do	do	Do.
19	P. Kratze	Near Fort Hamilton ave., Fort Hamilton, New York	4		do	do	Do.
19	M. Fowler	Near Franklin avenue, Fort Hamilton, New York	3		do	do	Do.
19	M. Monihans	do	1		do	do	Do.
19	M. Nunans	do	1		do	do	Do.
19	H. E. Bowers	Shore road, Fort Hamilton, New York	1		do	do	Do.
19	H. Despard	do	4		do	do	Do.
19	James Sicks	do	2		do	do	Do.
19	J. Robinson	Near Shore road, Fort Hamilton, New York	1		do	do	Do.
19	George Geletons	do	1		do	do	Do.
19	John Slater	do	1		do	do	Do.
20	A. Crapsey	do	5		do	do	Do.
20	J. Waters	do	8		do	do	Do.
20	Children's Aid Society	do	5		do	do	Do.
20	P. H. Platzgraf	do	5		do	do	Do.
20	Ex-Mayor Gunther	Shore road, Locust Grove, New York	1		do	do	Do.
20	W. Rempson	do	1		do	do	Do.
20	Dr. Spear	do	3		do	do	Do.
20	M. O'Brien	do	8		do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections on Long Island, New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.							
Aug. 20	George Shields	Shore road, Locust Grove, New Yoek	2		Good	Good	Rowland & Hawk.
20	George W. Hennings	do	3		do	do	Do.
20	C. Walch	do	1		do	do	Do.
20	C. Baker	do	1		do	do	Do.
20	F. Gillbay	3d avenue, Fort Hamilton, New York	1		do	do	Do.
20	F. Waltz	do	1		do	do	Do.
20	J. F. Troumey	Near 3d avenue, Fort Hamilton, New York	3		do	do	Do.
20	John McCarty	do	5		do	do	Do.
20	J. McGlin	do	4		do	do	Do.
20	John Murphy	Near 920th street, Fort Hamilton, New York	8		do	do	Do.
21	M. Van Sickle	Near Brighton Place, Fort Hamilton, New York	2		do	do	Do.
21	Mr. Conway	do	2		do	do	Do.
21	Mr. Fisher	Near Overton avenue, Fort Hamilton, New York	1		do	do	Do.
21	Unknown	On meadows near Coney Island, New York	2		do	do	Do.
21	J. L. Voorhees	Near Boulevard, Gravesend, New York	2		do	do	Do.
21	M. Vandevere	do	1		do	do	Do.
21	J. Bar	Near Shore road, New Utrecht, N. Y	1		do	do	Do.
21	J. F. Berry	On Bath road, New Utrecht, N. Y	2		do	do	Do.
21	M. Benson	do	1		do	do	Do.
21	J. S. Van Pelt	do	1		do	do	Do.
21	Jacob S. Van Pelt	do	1		do	do	Do.
21	J. Smith	do	1		do	do	Do.
21	Morris Hagan	do	9		do	do	Do.
21	W. Quigley	John Bennett's lane, New Utrecht, N. Y	3		do	do	Do.
21	John Bennett	do	3		do	do	Do.
21	P. Murphy	Near Shore road, near Fort Hamilton, New York	11		do	do	Do.
21	A. Evens	Road to Fort Hamilton, Gravesend, N. Y	2		do	do	Do.
21	Mr. Hagan	do	1		do	do	Do.
21	J. Lake	do	5		do	do	Do.
21	David Jones	do	3		do	do	Do.
21	R. Peters	Bath road, New York	1		do	do	Do.
23	F. R. Ryan	Union street, Brownsville, N. Y	35	1	do	do	Do.
23	T. McGee	Rockaway avenue, New Lots, N. Y	54	1	do	do	Do.
23	Geo. Leeblara	Near Rockaway avenue, New Lots, N. Y	26		do	do	Do.
23	Thomas Shager	do	2		do	do	Do.
25	L. Gimpels	Near West street, New Lots, NY.	19	1	Bad	do	Do.
25	H. Gimpels	do	8		Good	do	Do.
25	C. E. Whitson	Center street, New Lots, N. Y	26	6	Poor	Poor	Do.

25	H. V. Rice	Rockaway avenue, New Lots, N. Y	13		Good	Good	Do.
25	Mrs. Sparker	Rockaway avenue, Brownsville, N. Y	29		do	do	Do.
25	W. H. Ryan	Blake avenue, Brownsville, N. Y	10	2	Poor	do	Do.
27	J. Vandevere	Hunterfly road, New Lots, N. Y	1		Good	do	Do.
27	J. Kinson	do	1		do	do	Do.
27	J. Balstridge	New Lots road, New Lots, N. Y	6		do	do	Do.
27	M. Blindfolder	do	1		do	do	Do.
27	W. Lenangarten	do	1		do	do	Do.
27	Sam'l Raphlee	do	11		do	do	Do.
27	W. Raphlee	do	4		do	do	Do.
27	H. Raphlee	do	1		do	do	Do.
27	W. R. Glover	do	1		do	do	Do.
27	J. C. Van Sicklen	Bear street, East New York, N. Y	2		do	do	Do.
27	L. Dryell	do	4		do	do	Do.
27	F. Belachk	Vermont avenue, East New York, N. Y	1		do	do	Do.
27	Fred. Bustch	do	4		do	do	Do.
27	Adam Cranmor	New Jersey avenue, East New York, N. Y	6		do	do	Do.
27	P Hire	Sheffield avenue, East New York, N. Y	5		do	do	Do.
27	H. Trish	Georgia avenue, East New York, N. Y	5		do	do	Do.
27	Conrad Roberts	Broadway, East New York, N. Y	1		do	do	Do.
27	C. S. Sultze	do	1		do	do	Do.
27	H. Line	Sheffield avenue, East New York, N. Y	1		do	do	Do.
27	Ward Saddler	Pennsylvania avenue, East New York, N. Y	1		do	do	Do.
27	H. Beam	do	9		do	do	Do.
27	H. Shaffer	do	7		do	do	Do.
27	F. Huttenlocher	do	4		do	do	Do.
27	E. Hickman	Johnson avenue, East New York, N. Y	12		do	do	Do.
27	T. Collins	Oriander avenue, East New York, N. Y	12		do	do	Do.
27	P. Schrouss	Near East New York, N. Y	45		do	do	Do.
28	S. Berdell	New Lots road, East New York, N. Y	45		do	do	Do.
28	J. Van Sicklen	do	2		do	do	Do.
28	W. Hopkins	do	1		do	do	Do.
28	F. McCalaus	do	29		do	do	Do.
28	Geo. Denton	do	1		do	do	Do.
28	William Slater	do	1		do	do	Do.
28	S. W. Tar	do	1		do	do	Do.
28	Joseph Hagerman	do	1		do	do	Do.
28	P. Appolee	do	1		do	do	Do.
28	Jacob Appolee	do	2		do	do	Do.
28	Nicholas Appolee	do	1		do	do	Do.
28	John Myres	do	18		do	do	Do.
28	H. Gartleman	do	1	1	do	do	Do.
28	Geo. Moore	do	1		do	do	Do.
28	N. Shull	Shepherd's avenue, East New York, N. Y	3		do	do	Do.
28	Philip Lang	do	6		do	do	Do.
28	W. Hagerman	Monroe avenue, East New York, N. Y	1	1	do	Fair	Do.
28	Jacob Hire	Liberty avenue, East New York, N. Y	7		do	Good	Do.
28	Mrs. M. Hagan	Bay avenue, East New York, N. Y	5		Filthy	do	Do.
28	J. Ragany	Madison avenue, East New York, N. Y	2		Good	do	Do.
28	M. Rinkey	Baltic avenue, East New York, N. Y	3		do	do	Do.
28	Mrs. Hultz	Elder avenue, East New York, N. Y	2		do	do	Do.
28	Saml. Smith	New York avenue, East New York, N. Y	14		Very good	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections on Long Island, New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884. Aug. 28	J. Matt	Christopher avenue, East New York, N. Y	30	4	Good	Good	Rowland & Hawk.
28	J. W. Hewette	Liberty street, East New York, N. Y	28	2		do	Do.
28	M. Van Sythe	do	22	2	Very good	do	Do.
30	Mr. Lewis	East New York avenue, East New York, N. Y	1		Good	do	Do.
30	G. H. Burns	do	20	1	Very good	do	Do.
30	E. Rian	do	14		Good	do	Do.
30	G. A. Bennett	Stone avenue, East New York, N. Y	25		Very good	do	Do.
30	C. H. Rian	East New York avenue, East New York, N. Y	47		do	do	Do.
18	Sundry owners	Lots near Newtown Creek, Brooklyn, N. Y	71			do	Wm. B. E. Miller.
18	James Tufts	Near Catholic cemetery, Brooklyn, N. Y	18	2	Good	do	Do.
18	John Chambres	do	6		do	do	Do.
19	Wm. Heyer	193 Nostrand avenue, Brooklyn, N. Y	7		do	do	Do.
19	Arthur Miller	Bushwick avenue, Brooklyn, N. Y	4		do	do	Do.
19	James Staples	do			do	do	Do.
19	Martin Jones	do	23		do	do	Do.
20	Wm. Payne	Bedford and Fulton streets, Brooklyn, N. Y	1		do	do	Do.
20	Thomas Gorden	186 Kosciusko street, Brooklyn, N. Y	1		do	do	Do.
20	Nicholas Ehler	321 Marcy street, Brooklyn, N. Y	1		do	do	Do.
20	Sundry owners	do	44		do	do	Do.
21	Thomas Dugan	40 Stockholm avenue, Brooklyn, N. Y	5		do	do	Do.
21	J. Lerand	1071 Myrtle avenue, Brooklyn, N. Y	7		do	do	Do.
21	Margaret Nash	504 Quincy avenue, Brooklyn, N. Y	3		do	do	Do.
21	J. M. Phelps	899 De Kalb avenue, Brooklyn, N. Y	2		do	do	Do.
21	W. Breckhausen	149 Throop avenue, Brooklyn, N. Y	2		do	do	Do.
21	John Fox	192 Herkimer avenue, Brooklyn, N. Y	5		do	do	Do.
21	Charles Vandevere	153 Nostrand avenue, Brooklyn, N. Y	1		do	do	Do.
23	Wm. Casper	Maspeth and Kingman avenues, Brooklyn, N. Y	5		do	do	Do.
23	Andrew Shall	Wyckoff and Starr avenues, Brooklyn, N. Y	1		do	do	Do.
23	Wm. Shehan	184 Meserole avenue, Brooklyn, N. Y	12		Filthy	Fair	Do.
23	Anthony Shultz	Irving and Stockholm avenues, Brooklyn, N. Y			Good		Do.
23	Unknown	Stockholm avenue, Brooklyn, N. Y	21			Good	Do.
25	John Mingle	248 Varick avenue, Brooklyn, N. Y	1		Good	do	Do.
25	J. W. Layton	Evergreen avenue and Jacob street, Brooklyn, N. Y	31	1	do	do	Do.
25	Charles Highland	10 Furman avenue, Brooklyn, N. Y	1		do	do	Do.
25	Charles Brocker	Broadway and Conway street, Brooklyn, N. Y					Do.
26	John Lick	226 1st street, Brooklyn, N. Y	4		Filthy	Fair	Do.
26	Catharine Ducker	5 Agate street, Brooklyn, N. Y	4		do	do	Do.
26	Geo. A. Saffer	Prospect avenue continued, Brooklyn, N. Y	2		Good	Good	Do.
26	John Wilheim	24 Hamburg avenue, Brooklyn, N. Y	6		Fair	Fair	Do.

	26	Rudolph Shein	George avenue, Brooklyn, N. Y	6		Filthy	do	Do.
	26	Michael Derleth	16 Hamburg avenue, Brooklyn, N. Y	5		Fair	do	Do.
	26	Nicholas Letner	112 Prospect avenue, Brooklyn, N. Y	3		do	do	Do.
	26	Joseph Shick	26 Monteith avenue, Brooklyn, N. Y	1		do	do	Do.
	26	James Murphy	112 Palmetto avenue, Brooklyn, N. Y	4		Good	Good	Do.
	26	Geo. Kurhardt	97 Adams avenue, Brooklyn, N. Y	17		do	do	Do.
	28	Dr. McLean	272 Hudson avenue, Brooklyn, N. Y	1				Do.
	28	Geo. Kramer	34 Bogart avenue, Brooklyn, N. Y	5		Good	Good	Do.
	28	Joseph Fahling	93 Evergreen avenue, Brooklyn, N. Y	2		Filthy	Fair	Do.
	28	Ann Singley	Moore street, Brooklyn, N. Y	6		do	do	Do.
	28	Margaret Kiepp	318 Evergreen avenue, Brooklyn, N. Y	2		Good	Good	Do.
	28	Mr. Thornton	19 Richardson avenue, Brooklyn, N. Y	6		Fair	do	Do.
	28	James Fletcher	254 Withers avenue, Brooklyn, N. Y	3		Filthy	Fair	Do.
	28	Rosa Doyle	Morgan and Parker avenues, Brooklyn, N. Y	12		do	do	Do.
	29	John Sirus	105 Kingsland avenue, Brooklyn, N. Y	5		Good	Good	Do.
	29	Peter Kinney	102 Guernsey avenue, Brooklyn, N. Y	4		do	do	Do.
	29	Catharine McCanne	169 Elm avenue, Brooklyn, N. Y	6		do	do	Do.
	29	P. Kelly	Stockholm and Irving avenues, Brooklyn, N. Y	6		Filthy	Fair	Do.
	29	Louisa Eucker	Raymond near Elwell avenue, Brooklyn, N. Y	1		Fair	do	Do.
	29	P. Wright	68 Dobbins avenue, Brooklyn, N. Y	1		Good	Good	Do.
	29	Robt. McDonald	India and Oakland avenues, Brooklyn, N. Y	20	4	Fair		Do.
	30	John Whitehouse	Duryea and Evergreen avenues, Brooklyn, N. Y	15	2		Bad	Do.
	30	Max Kichemeyer	34 Moore avenue, Brooklyn, N. Y	2		Filthy		Do.
	30	J. C. Trader	Schauffler and Central avenues, Brooklyn, N. Y			Bad		Do.
	30	Nettie Benjamin	do			do		Do.
	30	Edward Hacker	do			do	Fair	Do.
	30	C. Finken	Covert near Central avenue, Brooklyn, N. Y	20	4	Fair	Good	Do.
	30	A. Dixon	Schauffler and Central avenues, Brooklyn, N. Y	13	2		do	Do.
Sept.	1	Mrs. Van Winkle	Coppes and Central avenues, Brooklyn, N. Y	28		Good	do	Do.
	1	Joseph Hartley	Crayson and Central avenues, Brooklyn, N. Y	8	5	do	do	Do.
	1	E. J. Mott	Vorhees street and Central avenue, Brooklyn, N. Y	37	4	do	do	Do.
	1	John Boyle	158 Dupont street, Brooklyn, N. Y	2		do	do	Do.
	1	D. Murphy	195 Dupont street, Brooklyn, N. Y	2		do	do	Do.
	1	Ellen Gallagher	189 Dupont street, Brooklyn, N. Y	2		do	do	Do.
	1	John O. Hanlan	197 Dupont street, Brooklyn, N. Y	3		do	do	Do.
	1	Robt. Curry	155 Green street, Brooklyn, N. Y	4		do	do	Do.
	1	James Hubbard	North Henry street, Brooklyn, N. Y	6		do	do	Do.
	1	Alexander Johnson	110 Van Cott street, Brooklyn, N. Y	8		do	do	Do.
	1	Conrad Lamb	Corner Van Cott and Russell streets, Brooklyn, N. Y	6		do	do	Do.
	1	Mr. McKenna	175 Green Point avenue, Brooklyn, N. Y	1		do	do	Do.
	1	Jane Thompson	289 Green Point avenue, Brooklyn, N. Y	3		do	do	Do.
	1	Winfield McGloine	89 Diamond avenue, Brooklyn, N. Y	4		do	do	Do.
	1	Conrad Rhode	36 Diamond avenue, Brooklyn, N. Y	1		do	do	Do.
	4	James O'Neil	57 Diamond avenue, Brooklyn, N. Y	2		do	do	Do.
	4	Wm. Shehan	162 Meserole avenue, Brooklyn, N. Y	12		do	do	Do.
	5	August Zimmerman	20 Thames avenue, Brooklyn, N. Y	7		Filthy	Fair	Do.
	5	Joseph Grassman	29 Thames avenue, Brooklyn, N. Y	5		do	do	Do.
	5	Eliza Bluer	23 Thames avenue, Brooklyn, N. Y	3		Good	Good	Do.
	5	Paul Hoffman	15 Grattan avenue, Brooklyn, N. Y	4		do	do	Do.
	6	Fred. Wolf	181 Trautman avenue, Brooklyn, N. Y	8		Fair	Fair	Do.
	6	Henry Josenhaus	167 Trautman avenue, Brooklyn, N. Y	4		Good	Good	Do.
	6	Wm. Goetz	57 Trautman avenue, Brooklyn, N. Y	2		do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections on Long Island, New York—Continued.

Date.	Name of owner or person in charge.	Location of stable and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884. Sept. 6	Sophia Bauer	31 Trautman avenue, Brooklyn, N. Y	2		Good	Good	Wm. B. E. Miller.
6	Gotter Keister	Ralph, near Evergreen avenue, Brooklyn, N Y	8		do	do	Do.
6	James Carroll	364 Trautman avenue, Brooklyn, N. Y	13	2	Fair	Fair	Do.
6	Fred'k Butzku	Trautman near Wyckoff avenue, Brooklyn, N. Y	23	2	Good	Good	Do·
6	Edmund Stirnn	do	5	2			Do.
6	Raphiel Brown	142 Larimer avenue, Brooklyn, N. Y					Do.
6	Adam Dietlin	129½ Cook avenue, Brooklyn, N. Y	1		Good	Good	Do.
2	J. Buffett	Near Union street, East New York, N. Y	43		do	do	Rowland & Hawk.
2	A. S. Bardell	Near East New York avenue, East New York, N. Y	31	5	do	do	Do.
2	A. Wanser	do	12	2	do	do	Do.
3	W. H. Rogers	Conklin avenue, near Canarsie, New York	2		do	do	Do.
3	P. Lewser	do	3		do	do	Do.
3	A. Deal	do	1		do	do	Do.
3	J. W. Rump	Canarsie road, New Lots, New York	1		do	do	Do.
3	H. Kryer	do	2		do	do	Do.
3	M. Keller	do	1		do	do	Do.
3	Austin Burdell	do	11		do	do	Do.
3	Mrs. Sullivan	do	1		do	do	Do.
3	D. and J. W. Vandevere	Mill road, near Canarsie, New York	7		d3	do	Do.
3	Albert Rice	Rockaway avenue, East New York, N. Y	1		do	do	Do.
3	Mrs. Stine	do	1		do	do	Do.
3	J. B. Brunchedy	do	1		do	do	Do.
3	J. Miller	Rockaway avenue, near Canarsie, N. Y	2		do	do	Do.
3	W. Murphy	do	1		do	do	Do.
3	Geo. Battler	do	1		do	do	Do.
3	W. Tryer	do	1		do	do	Do.
3	Joseph Smith	do	1		do	do	Do.
3	C. Minst	do	2		do	do	Do.
3	M. Cadi	do	1		do	do	Do.
3	D. Teman	do	3		do	do	Do.
3	J. Haddleback	do	7		Filthy	do	Do.
3	C. Egart	do	24		Very fine	Very fine	Do.
3	James Vitlgas	do	2		Good	Good	Do.
3	W. Hizer	do	1		do	do	Do.
4	Godslip Hickally	Barberry street, near Canarsie, N. Y	10		do	do	Do.
4	J. Hellagan	Van Sicklin avenue, East New York, N. Y	3		do	do	Do.
4	J. Blackwell	Barberry avenue, East New York, N. Y	11		do	do	Do.
4	Mrs. Obear	Madison street, East New York, N. Y	1		do	do	Do.

4	M. Haglan	do	1		do	do	Do.
4	J. Hire	Liberty street, East New York, N. Y	5		do	do	Do.
4	Vernica Shipps	do	3		do	do	Do.
4	P. Raplee	do	4		do	do	Do.
4	H. Hergerton	do	1		do	do	Do.
4	W. Douglass	do	1		do	do	Do.
4	E. V. W. Shaw	Atlanta avenue, East New York, N. Y	17		do	do	Do.
4	Thomas Grady	Elder avenue, East New York, N. Y	2		do	do	Do.
4	H. Brandwell	Jamaica plank road, Woodhaven, N. Y	15		do	do	Do.
4	Z. Brush	do	28	1	do	do	Do.
5	Joseph Lang	do	1		do	do	Do.
5	Frederick Frunger	Division street, Woodhaven, N. Y	1		do	do	Do.
5	M. Milander	do	1		do	do	Do.
5	Mrs. Susan Smith	do	6		do	do	Do.
5	Mr. Reiner	Jamaica plank road, East New York	2		do	do	Do.
5	J. J. Powell	Jamaica plank road, Woodhaven, N. Y	20		do	do	Do.
5	J. C. Schenk	do	1		do	do	Do.
5	A. C. Henry	do	51		do	do	Do.
5	H. Bauman	do	61	6	do	do	Do.
5	J. H. Smith	do	14	9	do	do	Do.
6	Mrs. Lyme	do	2	2	Fair	Fair	Do.
6	M. Bady	do	1		Good	Good	Do.
6	M. Platt	do	2		do	do	Do.
6	J. Van Sicklin	do	2		do	do	Do.
6	Lewis Fay	do	6		do	do	Do.
6	W. Kamman	do	1		do	do	Do.
6	T. Peffer	do	1		do	do	Do.
6	J. V. Latts	do	2		do	do	Do.
6	M. Lotts	do	1		do	do	Do.
6	J. Murs	do	14		do	do	Do.
6	B. J. Bryon	do	32	3	do	do	Do.
6	D. S. Lotts	Jamaica plank road, Jamaica, N. Y	12		do	do	Do.
6	J. L. Wycoff	do	1		do	do	Do.
6	M. McFraze	do	1		do	do	Do.
6	J. T. Suden	do	1		do	do	Do.
6	M. Caffield	Hail avenue, Woodhaven, N. Y	4		do	do	Do.
6	H. Copman	Cypress avenue, Woodhaven, N. Y	1		do	do	Do.
6	M. Gibins	Fulton avenue, Woodhaven, New York	2		do	do	Do.
6	M. Sall	Market street, Woodhaven, New York	3		do	do	Do.
6	S. J. Smith	do	29		do	do	Do.
6	John Lanher	Chestnut street, Woodhaven, New York	4		do	do	Do.
6	M. Gibbons	do	2		do	do	Do.
6	A. Monhawker	do	2		do	do	Do.
6	Walker Abner	do	1		do	do	Do.
6	Dan Douglas	Locust street, Woodhaven, New York	2		do	do	Do.
8	Fred Clump	Jamaica plank road, Jamaica, New York	27		do	do	Do.
8	M. Brucy	do	1		do	do	Do.
8	C. Bullman	do	1		do	do	Do.
8	M. Cinbit	do	5		do	do	Do.
8	M. Correll	do	2		do	do	Do.
10	C. Grace	do	1		do	do	Do.
10	S. Collins	do	4		do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections on Long Island, New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.							
Sept. 10	M. Carl	Jamaica plank road, Jamaica, N. Y	2		Good	Good	Rowland & Hawk.
10	Joseph Conklin	do	1		do	do	Do.
10	G. L. Fowler	do	6		do	do	Do.
10	John Stewart	do	1		do	do	Do.
10	F. W. Dunton	do	5		do	do	Do.
10	H. D. Burgin	do	1		do	do	Do.
10	Geo. Billman	do	1		do	do	Do.
10	Nicholas Corbin	do	9		do	do	Do.
10	James Walch	do	4		do	do	Do.
10	M. Winzire	do	2		do	do	Do.
10	J. Stillmire	Atlanta avenue, Jamaica, New York	7		do	do	Do.
10	Martin G. Johnson	Liberty street, Jamaica, New York	1		do	do	Do.
11	M. Maller	Near Williamsburg turnpike, Jamaica, N. Y	1		do	do	Do.
11	M. Ohare	do	1		do	do	Do.
11	Jacob Gunther	do	2		do	do	Do.
11	W. Wagner	do	1		do	do	Do.
11	Tom Kelley	Jamaica plank road, Jamaica, N. Y	1		do	do	Do.
11	C. Hench	do	1		do	do	Do.
11	P. Kelley	do	1		do	do	Do.
11	C. Warner	do	1		do	do	Do.
11	J. Dropbrinskey	do	1		do	do	Do.
11	A. Slaughtenburg	do	1		do	do	Do.
11	W. A. Jones	do	4		do	do	Do.
11	J. Mosin	do	1		do	do	Do.
11	M. Kelley	do	2		do	do	Do.
11	Geo. Nostrand	do	1		do	do	Do.
11	Geo. Andergre	do	2		do	do	Do.
11	E. Campion	do	2		do	do	Do.
11	A. A. DeGraw	do	35		do	do	Do.
11	Geo. Jones	do	21		do	do	Do.
11	C. Miller	do	1		do	do	Do.
11	P. Curigan	do	1		do	do	Do.
11	Geo. Kellow	do	1		do	do	Do.
11	Richard Neal	Near York avenue, Jamaica, N. Y	1		do	do	Do.
11	Dan. Smith	York avenue, Jamaica, N. Y	1		do	do	Do.
11	M. Skidmore	Near York avenue, Jamaica, N. Y	1		do	do	Do.
11		do	7		do	do	Do.
13	R. Connit	Near Beaver street, Jamaica, N. Y	6		do	do	Do.
13	A. McCormick	Jamaica plank road, Jamaica, N. Y	2		do	do	Do.

18	M. Nolan	Johnson avenue, Jamaica, N. Y	2		do	do	Do.
18	John Masterson	Near Boulevard, Jamaica, N. Y	1		do	do	Do.
18	A. Ford	Beaver street, Jamaica, N. Y	3		do	do	Do.
18	C. Ashenbach	do	1		do	do	Do.
18	C. Sultz	do	1		do	do	Do.
13	J. Shevers	do	2		do	do	Do.
13	M. Henry	do	2		do	do	Do.
13	P. Larkin	do	1		do	do	Do.
13		Catharine street, Jamaica, N. Y	7		ho	do	Do.
18	John Sweeney	do	3		do	do	Do.
13	C. Travers	do	1		do	do	Do.
13	H. Rutgers	Marrick road, Jamaica, N. Y	1		do	do	Do.
13	E. Signer	do	1		do	do	Do.
13	J. C. Hendricks	do	6		do	do	Do.
13	M. Feedmore	Union Hall street, Jamaica, N. Y	1		do	do	Do.
13	C. Clifton	do	1		do	do	Do.
13	M. Burgh	Douglas street, Jamaica, N. Y	5		do	do	Do.
13	Adam Martin	do	1		do	do	Do.
13	Mrs. Widerman	do	2		do	do	Do.
13	M. Mingins	do	3		do	do	Do.
13	Andrew Smith	South street, Jamaica, N. Y	2		do	do	Do.
13	August Baker	Rockaway road, Jamaica, N. Y	3		do	do	Do.
13	M. Armstrong	do	1		do	do	Do.
13	S. Sours	do	1		do	do	Do.
13	J. A. Suydam	do	3		do	do	Do.
13	M. Beeds	do	6		do	do	Do.
13	J. Smith	do	3		do	do	Do.
13	M. Davis	do	1		do	do	Do.
15	C. Hummer	do	2		do	do	Do.
15	Z. Hattie	do	2		do	do	Do.
15	J. W. Silver	do	1		do	do	Do.
15	M. Kempton	do	2		do	do	Do.
15	Dr. Morris	do	4				Do.
15	M. Duyea	do	1				Do.
15	J. Sneidicar	do	2				Do.
15	C. Nickolds	do	2				Do.
15	J. Studifud	do	3				Do.
15	P. Conklin	do	2				Do.
15	Jessie Williamson	do	1				Do.
8	Frank Evans	53 Ewen street, Brooklyn, N. Y	4				Wm. B. E. Miller.
8	Michael Greer	833 Flushing street, Brooklyn, N. Y	1		Bad	Bad	Do.
8	Mary Darly	7 Walton street, Brooklyn, N. Y	1		do	do	Do.
8	Frank Schwerin	167 McKibben street, Brooklyn, N, Y	2	1	Very bad	do	Do.
10	Thomas Schwerin	15 Graham street, Brooklyn, N. Y	2		Good	Good	Do.
10	F. E. Matts	216 Mott street, Brooklyn, N. Y	25		do	do	Do.
10	Wm. Lottz	258 Hopkins street, Brooklyn, N. Y	5		do	do	Do.
10	(Unknown)	Myrtle avenue, Brooklyn, N. Y	5	3	Fair	do	Do.
10	Samuel Retchie	331 Rutledge street, Brooklyn, N. Y	7				Do.
10	Abraham Hubpts	316 Rutledge street, Brooklyn, N. Y	6		Good	Good	Do.
10	F. Conners	151 North 4th street, Brooklyn, N. Y	2		Filthy	Fair	Do.
11	J. Muldoon	218 Wallabout street, Brooklyn, N. Y	2	1	Good	Good	Do.
11	Jos. Sillmer	48 Melrose street, Brooklyn, N. Y	3		do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections on Long Island, New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.							
Sept. 11	Andrew Quint	142 Melrose street, Brooklyn, N. Y	3		Good	Good	Wm. B. E. Miller.
11	Fred Schmelser	79 Melrose street, Brooklyn, N. Y	3		do	do	Do.
11	John Hoffman	65 Central avenue, Brooklyn, N. Y	5		Fair	do	Do.
18	August Bangle	155 Central avenue, Brooklyn, N. Y	7		Good	do	Do.
18	Peter Joyce	Brooklyn, N. Y	15				Do.
18	Jane Joyce	Brooklyn, N. Y					Do.
18	Bridget Barker	156 Hamburg avenue, Brooklyn, N. Y	1		do	do	Do.
19	Daniel Gerryman	152 Hamburg avenue, Brooklyn, N. Y	2		do	do	Do.
19	Michael Nader	138 Hamburg avenue, Brooklyn, N. Y	5		do	do	Do.
19	Mr. Bayer	79 Starr street, Brooklyn, N. Y	1		do	do	Do.
19	John Doyle	311 Union avenue, Brooklyn	1		do	do	Do.
20	Emil Markret	50 Prospect avenue, Brooklyn, N. Y	25	3	Filthy	Fair	Do.
20	Henry Neiderman	121 Jefferson avenue, Brooklyn, N. Y					Do.
20	Michael Quigley	198 Stockholm street, Brooklyn, N. Y	8		Filthy	Bad	Do.
20	John Schrock	27 Faust street, Brooklyn, N. Y	2		Good	Good	Do.
20	Joseph Wolf	73 Adams street, Brooklyn, N. Y	3		do	do	Do.
8	Mrs. Hay	Jamaica plank road, Jamaica, N. Y	3		do	do	Rowland & Hawk.
8	J. Dumpsey	do	1		do	do	Do.
8	J. Robinson	do	1		do	do	Do.
8	Frank Bradley	do	1		do	do	Do.
8	James Light	do	1		do	do	Do.
8	John Taph	do	2		do	do	Do.
8	J. B. Neppers	do	6		do	do	Do.
8	Lewis Mark	do	3		do	do	Do.
8	H. Kopian	Wyckoff avenue, Jamaica, N. Y	3		do	do	Do.
8	J. Vandevere	Woodhaven avenue, Jamaica, N. Y	2		do	do	Do.
8	C. Laug	do	1		do	do	Do.
8	M. Grassyard	do	2		do	do	Do.
8	Anthony Terrie	University Place, Jamaica, N. Y	1		do	do	Do.
8	H. Dion	Rockaway road, Jamaica, N. Y	10		do	do	Do.
8	H. Bauman	do	1		do	do	Do.
8	(Unkown)	North second street, Jamaica, N. Y	4		do	do	Do.
8	P. Hearman	do	4		do	do	Do.
8	John Croak	do	4		do	do	Do.
8	J. Chorus	Near University Place Jamaica, N. Y	1		do	do	Do.
8	M. Weeks	University Place, Jamaica, N. Y	1		do	do	Do.
8	Mrs. A. Billey	do	1		do	do	Do.
8	C. Miller	Benedict avenue, Jamaica, N. Y	10		do	do	Rowland & Hawk.

8	C. Roder	do	1		do	do	Do.
8	Alf. Brashimer	do	5		do	do	Do.
8	J. Shotts	Near South road, Woodhaven, N. Y	1		do	do	Do.
8	John Sterburg	South road, Woodhaven, N. Y	8		do	do	Do.
15	J. R. Williamson	Rockaway avenue, Jamaica, N. Y	1		do	do	Do.
15	J. Allen	do	1		do	do	Do.
15	John Lamberton	do	2		do	do	Do.
15	Geo. Abrams	do	1		do	do	Do.
15	William Powell	do	1		do	do	Do.
15	Mrs. Duley	do	1		do	do	Do.
15	John Amblener	Springfield road, Jamaica, N. Y	2		do	do	Do.
15	W. Foster	do	8		do	do	Do.
15	John Burdell	do	2		do	do	Do.
15	A. Sager	do	1		do	do	Do.
15	John Kensey	do	3		do	do	Do.
15	N. J. Higby	do	8		do	do	Do.
15	Sam. Mills	Mills lane, Jamaica, N. Y	1		do	do	Do.
15	Charles Thorn	do	1		do	do	Do.
15	Dan. Murry	do	1		do	do	Do.
15	J. J. Rider	do	1		do	do	Do.
15	John Betsis	do	1		do	do	Do.
15	Thom. Smith	do	1		do	do	Do.
16	J. T. Crusher	Van Wick street, Jamaica, N. Y	1		do	do	Do.
16	John Masters	Near Liberty street, Jamaica, N. Y	1		do	do	Do.
16	John Zinc	do	2		do	do	Do.
16	Unknown	do	2		do	do	Do.
16	H. Eldert	do	2		do	do	Do.
16		do	2		do	do	Do.
16	Hyman Overblast	Liberty street, Jamaica, N. Y	3		do	do	Rowland.
16	H. Johnson	do	1		do	do	Do.
16	Tom Manahan	do	2		do	do	Do.
16	Joe Griffin	do	1		do	do	Do.
16	Martin Johnson	do	1		do	do	Do.
16	M. VanWinkle	do	8		do	do	Do.
16	Joseph App	do	2		do	do	Do.
16	A. Shaffer	do	1		do	do	Do.
16	John Smith	do	8		do	do	Do.
16	John Wissinger	do	1		do	do	Do.
16	Philip Moelins	Centreville avenue, Jamaica, N. Y	2		do	do	Do.
16	J. Bechling	do	1		do	do	Do.
16	August Gesen	do	1		do	do	Do.
16	Jacob Boas	do	1		do	do	Do.
16	J. G. Bryer	do	1		do	do	Do.
16	Albert Famelds	do	1		do	do	Do.
16	Philip Grant	Aqueduct avenue, Jamaica, N. Y	1		do	do	Do.
16	William Rider	do	8		do	do	Do.
16	M. Dinton	do	1		do	do	Do.
16	Stephen Rider	do	1		do	do	Do.
16	C. Morris	do	1		do	do	Do.
16	J. G. Strang	do	4		do	do	Do.
16	Jacob Beiren	do	5		do	do	Do.
16	C. Shang	do	1		do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections on Long Island, New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.							
Sept. 16	John Conover	Aqueduct avenue, Jamaica, N. Y	2		Good	Good	Rowland.
16	Henry Low	Old South road, Jamaica, N. Y	1		do	do	Do.
16	W. Van Sickler	Hestoring Creek, Jamaica, N. Y	2		do	do	Do.
16	C. Hockhearst	Lincoln avenue, Jamaica, N. Y	8		do	do	Do.
16	A. Van Sickler	do	2		do	do	Do.
17	Gen. Swink	Fulton street, Jamaica, N. Y	3		do	do	Rowland & Hawk.
17	M. Larkins	do	10		do	do	Do.
17	John Ledwards	do	1		do	do	Do.
17	Rogers Conneff	Dublin street, Jamaica, N. Y	5		do	do	Do.
17	Mrs. Washnell	do	2		do	do	Do.
17	Mich. Nolan	Near Depot street, Jamaica, N. Y	3		do	do	Do.
17	John Smith	do	3		do	do	Do.
17	James Van Sickle	Lincoln street, Jamaica, N. Y	3		do	do	Do.
17	G. Griffen	do	2		do	do	Do.
17	M. Van Sickle	do	1		do	do	Do.
17	James P. Frederich	do	2		do	do	Do.
17	M. Blarly	do	1		do	do	Do.
17	Dan. Fredricks	do	1		do	do	Do.
17	John Flinn	Old South road, Jamaica, N. Y	1		do	do	Do.
17	B. Henderson	do	1		do	do	Do.
17	B. Bergen	do	7		do	do	Do.
17	W. H. Fredricks	do	2		do	do	Do.
17	Thom. D. Smith	do	1		do	do	Do.
17	Jacob White	do	1		do	do	Do.
17	M. Johenas	do	1		do	do	Do.
17	C. Fredricks	do	2		do	do	Do.
17	Amos Smith	do	2		do	do	Do.
17	John Higby	Mill road, Jamaica, N. Y	2		do	do	Do.
17	Geo. Way	do	1		do	do	Do.
17	James Francis	do	9		do	do	Do.
17	A. Keifer	do	2		do	do	Do.
19	Samuel Seaman	Jamaica plank road, Jamaica, N. Y	57	5 c	Filthy	Not good	Do.
19	W. H. Comes	do	28		Good	Good	Do.
19	Elbert Van Sise	do	26		do	do	Do.
19	M. Brennan	Locust avenue, Jamaica, N. Y	1				Do.
19	Dan. Douglass	do	2		Good	Good	Do.
19	A. S. Montfort	New Lots road, Jamaica, N. Y	42		do	do	Do.
20	William Smith	Leing avenue, Jamaica, N. Y	2		do	do	Do.

20	G. Gunther	do	3		do	do	Do.
20	J. Dich	do	3		do	do	Do.
20	G. H. Higby	Marrick road, Jamaica, N. Y	2		do	do	Do.
20	W. R. Higby	do	1		do	do	Do.
20	P. Bailey	do	2		do	do	Do.
20	C. Adams	do	1		do	do	Do.
20	J. R. Carpenter	do	4		do	do	Do.
20	H. W. Wright	do	3		do	do	Do.
20	C. H. Wood	do	1		do	do	Do.
20	J. Tilbury	do	2		do	do	Do.
20	A. Stoder	do	1		do	do	Do.
20	C. Thompson	do	1		do	do	Do.
20	M. L. Biersall	do	1		do	do	Do.
20	H. Piersall	do	1		do	do	Do.
20	W. F. Auguston	do	2		do	do	Do.
20	J. Duplevoice	do	2		do	do	Do.
20	Edward Siney	do	2		do	do	Do.
20	P. Van Sicklen	Springfield road, Jamaica, N. Y	2		do	do	Do.
20	J. Dibblewith	do	2		do	do	Do.
20	Mrs. Henderson	do	2		do	do	Do.
20	Frank Lotts	do	3		do	do	Do.
20	D. J. Perdy	do	1		do	do	Do.
20	D. & A. C. Ketchum	do	3		do	do	Do.
20	S. Fredricks	do	3		do	do	Do.
20	Cornelius Daemeter	do	3		do	do	Do.
20	John Ditmers	do	1		do	do	Do.
20	J. D. B. Sudam	do	2		do	do	Do.
20	W. Kraumevacher	do	3		do	do	Do.
20	J. F. Henderson	do	1		do	do	Do.
20	D. B. Heardison	do	1		do	do	Do.
20	H. Kemton	do	2		do	do	Do.
20	E. Hendrickson	do	1		do	do	Do.
20	F. Hardes	do	1		do	do	Do.
20	J. Townsend	do	1		do	do	Do.
20	J. Bailey	do	1		do	do	Do.
20	J. Townsend	do	1		do	do	Do.
20	P. Bailey	do	2		do	do	Do.
20	E. J. Henderson	do	2		do	do	Do.
20	J. Studdler	do	1		do	do	Do.
20	Joe Long	do	2		do	do	Do.
20	J. Cocklay	do	3		do	do	Do.
20	Francis Buck	do	2		do	do	Do.
20	C. Stealer	do	3		do	do	Do.
20	Martin Miller	do	3		do	do	Do.
20	W. G. Henderson	do	2		do	do	Do,
20	James Lotts	New York aveune, Jamaica, N. Y	1		do	do	Do.
20	John Smith	Jamaica plank road, Jamaica, N. Y	2		do	do	Do.
20	Edward Tylees	do	1		do	do	Do.
20	Samuel Kazine	do	2		do	do	Do,
20	Clause Myers	do	1		do	do	Do.
20	Charles Myres	do	2		do	do	Do.
20	J. L. Brinchoff	do	1		do	do	Do,

Investigation of contagious pleuro-pneumonia—Inspections on Long Island, New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.							
Sept. 20	M. Creed	Jamaica plank road, Jamaica, N. Y	1		Good	Good	Rowland & Hawk.
22	Henry Ellen	Hillside avenue, Jamaica	2		do	do	Do.
22	Mrs. Oldragey	do	1		do	do	Do.
22	M. Kesey	Blackstump road, Jamaica, N. Y	2		do	do	Do.
22	John Dehl	do	28		do	do	Do.
22	C. Iseman	Qualsome road, Jamaica, N. Y	4		do	do	Do.
22	Frank Cow	Grand street, Jamaica, N. Y	4		do	do	Do.
22	James Pines	Douglass street, Jamaica, N. Y	1		do	do	Do.
22	O. Jackson	Madison street, Jamaica, N. Y	7		do	do	Do.
22	J. Stillman	do	1		do	do	Do.
22	Thomas Bell	Willow street, Jamaica, N. Y	2		do	do	Do.
22	E. Bell	do	2		do	do	Do.
22	Dan. Hogan	do	2		do	do	Do.
22	J. McDonald	do	3		do	do	Do.
22	Jos. Walter	Jamaica street, Jamaica, N. Y	1		do	do	Do.
22	J. K. Van Sicklen	do	5		do	do	Do.
22	A. J. Van Sicklen	do	2		do	do	Do.
22	W. Voise	do	1		do	do	Do.
22	John Kizer	do	1		do	do	Do.
22	Henry Neal	do	1		do	do	Do.
22	H. Kesell	Flushing street, Jamaica, N. Y	4		do	do	Do.
22	Mrs. Vanderburg	do	1		do	do	Do.
22	G. Van Dine	do	2		do	do	Do.
12	Joseph Schleider	do	1		do	do	Do.
23	Godslip Voga	do	2		do	do	Do.
23	Mrs. F. Baer	do	1		do	do	Do.
23	M. Claim	do	2		do	do	Do.
23	M. Hartman	do	1		do	do	Do.
23	M. Tartman	do	2		do	do	Do.
23	J. Rendlond	do	2		do	do	Do.
23	J. A. Hagerman	do	2		do	do	Do.
28	C. Dixon	Flushing road, Flushing	1		do	do	Do.
23	C. Rider	do	1		do	do	Do.
23	H. Keeal	do	1		do	do	Do.
23	M. Ely	do	25		do	do	Do.
23	John Mower	Blackstump road, Flushing	17	8 c	Filthy	Not good	Do.
23	Thom. Connor	do	1		Good	Good	Do.
23	A. Richard	do	10		do	do	Do.

23	John Hunt	do	1		do	do	Do.
23	John Diskle	do	3		do	do	Do.
23	J. Bophon	do	1		do	do	Do.
23	F. Ferrig	do	1		do	do	Do.
23	W. Hisslin	do	10		do	do	Do.
24	W. A. Willis	Jericho avenue, Mineola	1		do	do	Do.
24	T. Tredwell	do	3		do	do	Do.
24	J. H. Andros	do	1		do	do	Do.
24	Geo. Allen	do	3		do	do	Do.
24	H. C. Woodnut	do	3		do	do	Do.
24	R. H. Robins	do	4		do	do	Do.
24	A. C. Woodnut	Mineola	1		do	do	Do.
24	J. McCoy	do	4		do	do	Do.
24	S. Smith	do	4		do	do	Do.
24	A. W. Wilder	Woodbury	2		do	do	Do.
24	Scadder & Townsend	Glentear	1		do	do	Do.
24	S. M. Tytus	Glen Cove	1		do	do	Do.
24	H. Riddy	Merrick	1		do	do	Do.
24	Townsend Drock	Locust Valley	1		do	do	Do.
24	F. Japer	Rockville Center	1		do	do	Do.
24	A. Belmont	Babylon	3		do	do	Do.
24	W. P. Douglass	Douglasston	5		do	do	Do.
24	R. E. Dietz	Hempstead	1		do	do	Do.
24	James Bailey	do	1		do	do	Do.
24	J. Van Schick	do	1		do	do	Do.
24	J. W. Barnum	do	6		do	do	Do.
26	M. Alburton	Hempstead road, Mineola	2		do	do	Do.
26	Mr. Platt	do	1		do	do	Do.
26	Thomas Callias	do	1		do	do	Do.
26	Jacob Bergen	do	2		do	do	Do.
26	A. Howiter	do	4		do	do	Do.
26	Lewis Rasick, jr	do	2		do	do	Do.
26	Mr. Sherman	do	2		do	do	Do.
26	A. Stockholm	do	6		do	do	Do.
26	Joe Grult	do	1		do	do	Do.
26	John Parker	do	2		do	do	Do.
26	W. H. Kinsey	do	2		do	do	Do.
26	W. C. Valentine	do	1		do	do	Do.
26	W. Clemp	do	1		do	do	Do.
26	W. Raner	do	1		do	do	Do.
26	Fred. Dunton	do	4		do	do	Do.
26	Isaac Bailey	do	1		do	do	Do.
26	Unknown	do	2		do	do	Do.
26	Alex. Bates	do	1		do	do	Do.
26	John Cole	do	2		do	do	Do.
26	M. Frost	do	1		do	do	Do.
26	James Kelley	Clinton avenue, Mineola	7		do	do	Do.
26	M. Mitchell	do	8		do	do	Do.
26	Joe Buff	do	1		do	do	Do.
26	J. L. Bergen	Jericho avenue, Mineola	1		do	do	Do.
26	C. Hoffman	do	2		do	do	Do.
26	John Weeks	do	10		do	do	Do.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.							
Sept. 26	Mr. Levy	Rosiyn road, Mineola	150	......	Good	Good	Roland & Hawk.
29	Mrs. Ginder	Wyckoff avenue, Ridgewood	25	......	do	do	Do.
29	C. Heroveiler	Worth street, Ridgewood	8	......	do	do	Do.
29	A. Lichtenheidt	Green street, Ridgewood	10	......	Bad	do	Do.
29	Barney Linger	do	19	......	do	Bad	Do.
30	Philip Hubbert	Jericho avenue, Ridgewood	2	......	Good	Good	Do.
30	M. L. Orrison	Jericho avenue, Mineola	1	......	do	do	Do.
30	M. Woodnut	do	4	......	do	do	Do.
30	M. Smith	do	6	......	do	do	Do.
30	C. Weeks	do	4	......	do	do	Do.
30	H. B. Wilson	do	4	......	do	do	Do.
30	R. Valentine	do	25	......	do	do	Do.
30	Geo. Thorn	do	3	......	do	do	Do.
30	Thom. White	do	25	......	do	do	Do.
30	Sam. Armstrong	do	4	......	do	do	Do.
30	Joseph Armstrong	do	8	......	do	do	Do.
30	Sam. Keaty	do	2	......	do	do	Do.
30	Ben. Smith	do	1	......	do	do	Do.
30	M. Dightman	do	2	......	do	do	Do.
30	John McCartzer	do	1	......	do	do	Do.
30	M. Walsh	do	2	......	do	do	Do.
30	M. McCan	do	2	......	do	do	Do.
30	Gill Foy	do	1	......	do	do	Do.
30	Augusthus Dinther	do	5	......	do	do	Do.
30	Austin Dinther	do	2	......	do	do	Do.
30	W. H. Weeks	do	7	......	do	do	Do.
Oct. 1	Mrs. O'Farrell	Catharine street, Ridgewood	3	......	do	do	Do.
1	Unknown	Myrtle avenue, Ridgewood	8	......	do	do	Do.
1	Mrs. C. Denton	do	2	......	do	do	Do.
1	Charles Chopping	do	1	......	do	do	Do.
1	Henry Ceb	do	1	......	do	do	Do.
1	J. Jaskive	do	90	......	do	do	Do.
1	F. Dingerson	do	31	......	do	do	Do.
2	Timothy Shea	7th street, Hunter's Point	9	......	do	Bad	Do.
2	R. Vail	do	5	......	do	Good	Do.
2	John Mahamery	do	10	......	do	do	Do.
2	M. Grames	4th street, Hunter's Point	2	......	do	do	Do.
2	S. Sculley	Ferry street, Hunter's Point	3	......	do	do	Do.

2	E. Brogran	Jackson avenue, Hunter's Point	11		do	do	Do.
2	C. Nulty	do	2		do	do	Do.
2	Thom. Saidy	do	2		do	do	Do.
2	M. Donleys	do	1		do	do	Do.
2	F. McSpirit	Hunter's Point road	1		do	do	Do.
2	M. McCurdy	do	2		do	do	Do.
2	J. Allen	do	1		do	do	Do.
2	M. Doudan	do	3		do	do	Do.
2	J. Mahoney	do	10		do	do	Do.
2	M. Short	8th street, Hunter's Point	12		do	do	Do.
2	J. Shroder	7th street, Hunter's Point	5		do	do	Do.
2	M. Stevenson	do	1		do	do	Do.
2	John Ohanlan	Dupont street, Hunter's Point	5		do	do	Do.
8	Sam. Gorden	Green Point avenue, Blissville	138	16 1 *a*		Fair	Do.
8	Mike Clarency	Newtown road, Blissville	4	3 1 *a*		Bad	Do.
6	James Flarily	Woodside avenue, Blissville	8			Good	Do.
6	Mr. Powers	do	1			do	Do.
6	John Holly	do	6			do	Do.
6	M. Myres	do	36			do	Do.
6	Alfred Horn	Skillman avenue, Blissville	6			Good	Do.
6	Casper Becto	Green Point avenue	12	1 *a*			Do.
6	James Dunn	East 3d street, Long Island City	16			Good	Do.
6		In woods, Green Point avenue, Blissville		2 *a*			Do.
7	Philip Sebitte	Green Point avenue, Blissville	13	2 *a*	Filthy	Fair	Do.
7	G. Gorder	do	140		Good	Good	Do.
7	Casper Berton	do	13	2 *a*	Filthy	Bad	Do.
8	Mrs. Rose White	do	30	2 *a*	Good	Good	Do.
8	M. Myers	do	80		do	do	Do.
8	J. S. Kansattle	do	30	3	Poor	Fair	Do.
8	L. T. Stevenson	do	33	12	Filthy	Bad	Do.
8		Hill street, Blissville	1		Good	Good	Do.
9	Geo. Gosman	do	1		do	do	Do.
9	D. Hartgrove	do	1		do	do	Do.
9	F. Myres	Laurel Hill	36	12	Filthy	Bad	Do.
9	Mrs. Murphy	Washington avenue, Laurel Hill	8		Good	Good	Do.
9	W. Delvassa	do	2		do	do	Do.
9	J. McCullough	do	22	1	do	do	Do.
10	A. Spades	Shell road, Laurel Hill	17	5	Filthy	Fair	Do.
10	G. Hary	Water avenue, Laurel Hill	2		Good	Good	Do.
10	A. Claypole	do	2		do	do	Do.
10	W. M. Smith	Laurel Hill	9	9	Bad	Bad	Do.
10	John Dobbins	Newtown	27		Good	Good	Do.
10	F. Gerhard	do	17	5			Do.
10	M. Vals	do	1		Good	Good	Do.
10	M. Flatts	do	2		do	do	Do.
10	M. Cummings	do	1		do	do	Do.
13	Kate Klock	14th avenue, Astoria	8		do	do	Do.
13	Henry Erett	Broadway, Astoria	5		do	do	Do.
13	Augustus Albright	do	14		do	do	Do.
13	M. Hussettman	do	6		do	do	Do.
13	Christopher Tomsey	18th street, Astoria	16		do	do	Do.
13	James Tulley	do	1		do	do	Do.

Date.		Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.								
Oct.	13	Miller	Broadway, Astoria	1		Good	Good	Rowland & Hawk.
	13	John Bonmore	Woodside road, Woodside	40		do	do	Do.
	13	Richard Degran	do	84		do	do	Do.
	13	Timothy Sley	do	9		do	do	Do.
	14	Patrick Mackentire	Astoria	24		do	do	Do.
	14	John Blay	do	11		do	do	Do.
	14	M. Swensburg	do	3		do	do	Do.
	14	Timothy Carney	do	13		do	do	Do.
	14	John O. Keef	do	28		do	do	Do.
	14	M. Stiners	do	3		do	do	Do.
	14	M. Teeney	do	28		do	do	Do.
	14	M. Snider	do	5		do	do	Do.
	14	Mrs. Hedgewood	do	2		do	do	Do.
	15	Samuel Laular	4th street, Ravenswood	8		do	do	Do.
	15	Patrick Harty	15th street, Ravenswood	1		do	do	Do.
	15	Unknown	do	6		do	do	Do.
	15	Thomas Dodles	do	1		do	do	Do.
	15	J. D. Smith	do	1		do	do	Do.
	15	Unknown	do	11		do	do	Do.
	15	C. Witt	Ravenswood	3		do	do	Do.
	15	M. Smith	Hunter's Point	1		do	do	Do.
	15	M. Hopkins	do	1		do	do	Do.
	15	Samuel Ragany	do	4		do	do	Do.
	15	J. S. Smith	do	1		do	do	Do.
	15	Mrs. Clifford	do	1		do	do	Do.
	15	Terrence Cosgrove	Broadway, Hunter's Point	4		do	do	Do.
	15	Mrs. A. Walsh	Hancock avenue, Hunter's Point	4		do	do	Do.
	15	Mrs. B. Walsh	Vernon avenue, Astoria	4		do	do	Do.
	15	Frank Clark	do	5		do	do	Do.
	15	John Murphy	do	8		do	do	Do.
	15	Unknown	Broadway, Astoria	14		do	do	Do.
	15	M. Rost	Vernon avenue, Astoria	1		do	do	Do.
	15	John Mahoney	8th street, Astoria	8		do	do	Do.
	15	Richard Vail	7th street, Astoria	4		do	do	Do.
Sept.	22	Mrs. Catharine Mooney	Oakland avenue and India street, Brooklyn	17	4a	do	do	Wm. B. E. Miller.
	25	Mrs. Julia Gans	Cor. Evergreen ave. and Convent street, Brooklyn	17	1	do	do	Do.
Oct.	7	John Derwin	Cypress Hills road, Brooklyn	5	2	Fair	Fair	Do.
	14	Mrs. Derleth	16 Hamburg street, Brooklyn	5	2	do	do	Do.

	14	Mrs. Devine	Marion street, Brooklyn	26	8 1e			Do.
	16	John Hazard	Academy street, near Astoria	1			Good	Rowland & Hawk.
	16	John Blair	do	12			do	Do.
	16	William Woordel	do	8			do	Do.
	16	John Wander	do	8	1		Fair	Do.
	16	Christian Dampman	Washington street, Astoria	4			Good	Do.
	16	M. Dey	do	2			do	Do.
	16	John Fundershire	do	1			do	Do.
	16	Herman Wilkimber	do	1			do	Do.
	16	Geo. Petrick	Third avenue, Astoria	4			do	Do.
	16	Mrs. Redline	do	3			do	Do.
	16	Mrs. Kizer	Astoria	2			do	Do.
	16	James Curtis	Cinbelia street, Astoria	2			do	Do.
	16	Mary Tyrany	Flushing avenue, Astoria	3			do	Do.
	16	Barny Keegan	do	8			do	Do.
	16	Morris Brady	do	7			do	Do.
	16	Mrs. McGoss	Near Flushing avenue, Astoria	12		Good	do	Do.
	16	Michael Atley	do	2			do	Do.
	16	John Burns	do	3			do	Do.
	16	M. C. Hallet	Grand street, Astoria	2			do	Do.
	16	Thomas Reeves	do	3			do	Do.
	16	Mr. Teeney	do	7			do	Do.
	16	Mr. O'Connor	Near Grand street, Astoria	8			do	Do.
	16	William Kempt	Grand street, Astoria	2			do	Do.
	16	J. Hobert	4th avenue, Astoria	1			do	Do.
	17	Frank Breman	Bradford street, Astoria	4			do	Do.
	17	John Smith	4th street, Astoria	4			do	Do.
	17	Matthew Nugent	Near Bradford street, Astoria	2			do	Do.
	17	M. Blinter	do	1			do	Do.
	17	Mrs. Howell	do	1			do	Do.
	17	M. Walsh	do	6			do	Do.
	17	M. Gardner	do	4			do	Do.
	17	Mrs. Hay	Flushing avenue, Astoria	1			do	Do.
	17	M. Herhart	Near Flushing avenue, Astoria	5			do	Do.
	17	Unknown	do	3			do	Do.
	17	Rhinehart Lebeck	do	8			do	Do.
	17	Laurence Klane	Hoyt street, Astoria	12			do	Do.
	17	Mrs. C. Fingley	Raply street, Astoria	12			do	Do.
	17	M. Gablet	Near Raply street, Astoria	2			do	Do.
	17	Mrs. Ward	Near Flushing avenue, Astoria	1			do	Do.
	17	Jessie Montare	Near St. John's street and St. John's Place	3			do	Do.
	17	M. Manahan	do	3			do	Do.
	17	Mrs. M. Bleedy	do	10			do	Do.
	17	M. Fish	do	1			do	Do.
	17	Mr. Spultz	do	1			do	Do.
Oct.	17	Thomas McCarty	St. John's Place	3			do	Do.
	17	Mrs. Creden	do	4			do	Do.
	17	Henry Hogate	do	1			do	Do.
	17	Catharine Duram	Grand street, Astoria	3			do	Do.
	17	C. Appleton	Main street, Astoria	1			do	Do.
	21	J. C. Jackson	Bowerybay road, Woodside	2			do	Do.
	21	Frank Weaver	do	4			do	Do.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884. Oct. 21	Anthony Holman	Bowery road, Woodside	4			Good	Rowland & Hawk.
21	A. Busbey	do	2			do	Do.
21	John Shank	do	1			do	Do.
21	M. Coobooler	Woodside street, Woodside	1			do	Do.
21	J. Doughtre	Bowerybay road, Woodside	2			do	Do.
21	Henry Harmsey	15th street, New Town	21			do	Do.
21	Frank Beck	do	1			do	Do.
21	Follentine Gedford	do	2			do	Do.
21	M. Spires	15th street, Astoria	3			do	Do.
21	Mr. Albrock	South street, Astoria	12			do	Do.
21	J. L. Townsand	Roslyn	52			do	Do.
21	William Sistler	South street, Astoria	9			do	Do.
21	Martin Hause	South street, Woodside	1			do	Do.
21	John Myers	Flushing avenue, Woodside	6			do	Do.
21	M. Jager	do	3			do	Do.
21	J. Sleckline	do	1			do	Do.
21	M. Monker	do	8			do	Do.
21	H. S. Hanson	Bowerybay road, Woodside	11			do	Do.
21	F. Kelling	Woodside	59			do	Do.
23	Martin Wise	Near 5th street, Woodside	8			do	Do.
23	Henry Burges	do	2			do	Do.
23	John Adams	do	2			do	Do.
23	M. Chappell	Newtown	2			do	Do.
23	William Cameron	do	2			do	Do.
23	M. Warsmall	do	10			do	Do.
23	Oscar McCabe	do	1			do	Do.
23	James Daton	do	7			do	Do.
23	Henry Bosman	do	10			do	Do.
23	M. Dwyer	do	6			do	Do.
23	Mrs. Hogan	Charlotteville	2			do	Do.
23	Mr. Marks	do	1			do	Do.
23	Mr. Wricets	do	1			do	Do.
23	Mr. Waterman	do	10			do	Do.
23	Adam Dengler	do	2			do	Do.
23	H. Oldricht	do	2			do	Do.
23	M. Patricht	do	3			do	Do.
23	M. Bender	do	2			do	Do.
23	Mrs. Link	do	4			do	Do.

Oct.	23	Mrs. Rinker	do	1			do	Do.
	23	Mr. Busby	do	8			do	Do.
	23	Henry Harms	do	17			do	Do.
	23	M. Meswick	do	2			do	Do.
	23	M. Snider	do	1			do	Do.
	23	J. Mysinger	do	2			do	Do.
	23	Laurence Redline	Newtown	2			do	Do.
	23	M. Fleckerstine	do	2			do	Do.
	33	J. Walsh	do	6			do	Do.
	24	Mrs. Rosendale	Ridgewood	20			do	Do.
	24	Peter Bamboon	do	14			do	Do.
	24	H. Dermis	do	10			do	Do.
	24	H. E. Burdell	do	28			do	Do.
	24	Jacob Beman	do	17			Good	Do.
	24	William Verrick	do	11			do	Do.
	24	Mrs. Griffin	do	3			do	Do.
	24	Cornelius Van Brum	Brunnett street, Ridgewood	9			do	Do.
	24	Mrs Davis	Ridgewood	2			do	Do.
	25	John Roman	Out from Ridgewood	31			do	Do.
	25	Jacob Munson	do	3			do	Do.
	25	John Goir	do	12			do	Do.
	25	D. Homire	do	37			do	Do.
	25	M. Deschler		29			do	Do.
	27	J. K. Loble	Maspeth	7			do	Do.
	27	Michael Green	Maspeth Place, Maspeth	24			do	Do.
	27	Garrick Ferman	do	3			do	Do.
	27	John Gennett	do	26			do	Do.
	27	John Sahr	Brood street, Maspeth	31			do	Do.
	27	M. C. Whilt	5th street, Maspeth	2			do	Do.
	27	Geo. Heauff	5th street, Millonia	24	1		do	Do.
	27	Jacob Blank	Newtown	7			do	Do.
	27	Geo. Feak	do	5			do	Do.
	28	Henry Easer	Flushing avenue, Middle Village	20			do	Do.
	28	H. Granner	do	1			do	Do.
	28	Geo. Hubbs	do	104	5		do	Do.
	28	Ernest Hudge	do	1			do	Do.
	28	Edward Shewech	Middle Village	4		Not good	Not good	Do.
	28	Cornelius Herber	Flushing avenue, Middle Village	13		Good	do	Do.
	28	H. Myers	Middle Village	1		Very good	Very good	Do.
	29	H. Hubbs	Flushing avenue, Middle Village	59		do	do	Do.
	29	Rupert Dempsey	Middle Village	3			Good	Do.
	29	Geo. Mott	do	27	1		do	Do.
	29	Frank Eller	Flushing avenue, Newtown	2			do	Do.
	29	Charles Berdell	Newtown	80		Very good	Very good	Do.
	29	C. E. Van Lise	do	47			Good	Do.
	29	L. Wild	do	6			do	Do.
	29	P. Wycoff	do	2			do	Do.
	31	M. Rosenberg	Winfield avenue, Newtown	60			do	Do.
	31	Aluton Reger	do	4			do	Do.
	31	Peter Ule	do	1	—		do	Do.
	31	J. Hammon	do	1			do	Do.
	31	John Fermon	Middle Village	1			do	Do.

Investigation of contagious pleuro-pneumonia—Inspections on Long Island, New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.							
Oct. 31	Frank Moran	Middle Village	1			Good	Rowland & Hawk.
31	H. Houselbean	do	1			do	Do.
31	Frank Bower	Fresh Ponds	1			do	Do.
31	Henry Bardy	do	1			do	Do.
31	Joe Miller	do	1			do	Do.

INSPECTIONS ON STATEN ISLAND, NEW YORK.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
July 24	Pat. Corley	Sarahann street, Middleton	2		Middling	Not very good	William Rose.
24	J. Cupps	Hannah street, Middleton	11		Good	Good	Do.
24	McO'Brien	do	4	1	do	do	Do.
24	G. Gallagher	Swann street, Middleton	11		do	do	Do.
25	Mrs. Morris	Sarahann street, Middleton	15		Poor	Middling	Do.
25	Mrs. Hallon	Back street, Middleton	4		Good	Good	Do.
25	Pat. Conlon	do	11		do	do	Do.
25	Tim. Foley	Swan street, Middleton	8		Poor	Middling	Do.
26	Robert Hara	Sarahann street, Middleton	4		Good	Good	Do.
26	Mr. Steel	Richmond road, Middleton	11		do	do	Do.
26	Widow Moffatt	Feidler's Park, Middleton	4		do	do	Do.
26	Widow Davis	Hannah street, Middleton	3		Middling	do	Do.
28	Herman Soutas	Richmond road, Middleton	4		Good	do	Do.
28	Peter Van Pelt	St. Paul avenue, Middleton	7		do	do	Do.
28	Mr. Dolan	McKeon street, Middleton	7		Filthy	Fair	Do.
28	Mrs. Lew	Broad street, Middleton	3		Poor	Poor	Do.
28	Capt. Winters	Bay street, Middleton	5		Good	Good	Do.
28	August Horeman	Prospect street, Middleton	3		do	do	Do.
29	D. R. Hitchcock	Richmond road, Middleton	4		do	do	Do.
29	Mrs. Lastrange	Quinn street, Middleton	5		Poor	Poor	Do.
29	John Clifford	St. Paul avenue, Middleton	23		Middling	Fair	Do.
30	Wm. B. Duncan	Duncan's Hill, Middleton	15		Good	Good	Do.
30	J. Johnson	Grymes' Hill, Middleton	3		do	do	Do.
30	Mr. Lawrence	Eddy street, Middleton	3		do	do	Do.
30	J. J. Sisco	Richmond road, Middleton	18		do	do	Do.
31	Gen. W. G. Ward	Ward's Hill, Middleton	8		do	do	Do.
31	J. H. Vanderbilt	Vanderbilt Hill, Middleton	9		do	do	Do.

31	W. S. Nicholas	do	8		do	do	Do.
31	E. Concord	Duncan Hill, Middleton	9		do	do	Do.
5	C. King	Ward's Hill, Middleton	3		do	do	Do.
5	Geo. Meyer	Targee street, Middleton	6		Normal	Normal	Do.
5	Geo. Bechman	Gordon street, Middleton	28		do	do	Do.
5	Zeph Hipp	do	8		Poor	Poor	Do.
5	Mrs. Lynch	do	4		do	do	Do.
5	John Newhart	do	8		Normal	Normal	Do.
5	Chas. Huger	Richmond road, Middleton	8		Good	Good	Do.
5	T. W. Bayard	do	2		Normal	Normal	Do.
5	Jas. McNamee	Grymes' Hill	8		do	do	Do.
5	Gen. Gourdan	Middleton	2		do	do	Do.
6	Pat. McNulty	Gordon street, Middleton	4		do	do	Do.
6	Richard Considine	do	2		do	do	Do.
6	Luis Lichnstein	Broad street, Middleton	4		do	do	Do.
6	Mrs. Thompson	do	8		do	do	Do.
6	Mrs. Reinart	do	8		do	do	Do.
6	Mrs. McAnney	do	8		do	do	Do.
6	E. A. Moore	Richmond road, Middleton	8		do	do	Do.
6	Jas. Durkin	Varian street, Middleton	2		do	do	Do.
6	Mrs. T. Morgan	do	7		do	do	Do.
6	George Bechtel	Boyd's Hill, Middleton	2		do	do	Do.
6	Joseph Rubsam	Wright street, Middleton	2		do	do	Do.
6	Chas. Seaman	Brock street, Middleton	8		do	do	Do.
6	Mr. Halberman	Harrison street, Middleton	5		do	do	Do.
6	John D. Dix	Vanderbilt avenue, Middleton	2		do	do	Do.
7	Widow's Home	Bay street, Middleton	2		do	do	Do.
7	Fred. Durve	Osgood avenue, Middleton	23		do	do	Do.
7	Jas. Ferguson	Targee street, Middleton	4		do	do	Do.
7	John Harbung	Paxter street, Middleton	8		do	Poor	
7	Andrew Schmeiser	Antone Lane, Middleton	6	1	Poor	Normal	Do.
7	Chas. Scheumacher	Richmond road, Middleton	8		Normal	do	Do.
7	Chas. Bischoff	do	8		do	do	Do.
6	Michel Dwyer	do	8		do	do	Do.
8	John Zimmers	Gordon street, Middleton	6		do	do	Do.
8	H. Baxter	Targee street road, Middleton	2		do	do	Do.
8	J. Ratshot	Targee street, Middleton	2		Poor	Poor	Do.
8	J. McGuire	Osgood avenue, Middleton	6	*1	do	do	Do.
8	Mrs. Hager	Targee street, Middleton	4		Normal	Normal	Do.
8	Thos. Dunn	Osgood avenue, Middleton	7	1a 1c	do	do	Do.
8	Fred. Sonneman	do	12		do	do	Do.
7	Mrs. Miller	do	2		do	do	Do.
12	Mrs. Baum	Richmond road, Middleton	2		do	do	Do.
12	John McCaffrey	do	2		Poor	Poor	Do.
12	J. W. Egbert	do	8		Normal	Normal	Do.
12	Philip Schroeder	Vanderbilt avenue, Middleton	2		do	do	Do.
12	Mrs. Wilson	Osgood avenue, Middleton	2		Poor	Poor	Do.
12	John Grechling	do	4		do	do	Do.
12	A. Gronsel	do	8		Normal	Normal	Do.
12	Patt Ellmeyer	Burton Place, Middleton	8		Poor	Poor	Do.

*Dead.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884. Aug. 12	Jas. Clark	Cedar street, Middleton	8		Poor	Poor	William Rose.
12	Patt. Hannah	Gordon street, Middleton	4		do	do	Do.
12	John Drummond	do	4		do	do	Do.
12	Mrs. Baisley	do	2		Normal	Normal	Do.
12	Mrs. Breman	Targee street, Middleton	3		Poor	Poor	Do.
12	Geo. Hogan	do	6		do	do	Do.
13	Mrs. Bout	do	2		do	do	Do.
13	Charles Clegg	Center street, Southfield, N. Y	8		Normal	Normal	Do.
13	Fred. Bachman	Willow avenue, Southfield, N. Y	8		do	do	Do.
13	Philip Brady	do	4		do	do	Do.
13	Julius Fleischman	Willow avenue, Southfield, N. Y	5		do	do	Do.
13	Joseph Haltenmeyer	Railroad bridge, Southfield, N. Y	4	1 a	do	do	Do.
13	Max. Sight	St. Mary's avenue, Southfield, N. Y	138		Filthy	Poor	Do.
14	Dennis McCarthy	do	13		do	do	Do.
14	Louis Schwartze	do	25		Poor	do	Do.
14	Mrs. Camel	Herd street, Southfield, N. Y	3		Normal	Normal	Do.
14	Mrs. Burns	do	5		do	do	Do.
14	Mrs. Gorman	do	10		do	do	Do.
14	Mrs. Deg	do	2		do	do	Do.
14	Mrs. McNamara	do	5		do	do	Do.
14	Mrs. O'Brien	do			do	do	Do.
15	James Marks	1st and St. John's streets, Southfield, N. Y	3		do	do	Do.
15	J. Dunster	do	2		do	do	Do.
15	Mrs. Lagrange	do	2		do	do	Do.
15	Gus Conner	do	3		do	do	Do.
15	Mike Murray	2d and St. John's streets, Southfield, N. Y	6		do	do	Do.
15	Mrs. Cummings	do	1		do	do	Do.
15	Mrs. Stell	do	8		do	do	Do.
15	George Hodges	Jews' buildings, Southfield, N. Y	4		do	do	Do.
15	Gotleb Henney	Willow avenue, Southfield, N. Y	4	1	do	do	Do.
15	John G. Vaughn	do	1		do	do	Do.
15	Antone Meacci	do	2		do	do	Do.
15	Mr. Butler	New York avenue, Southfield, N. Y	2		do	do	Do.
15	Captain King	do	5		do	do	Do.
15	Mr. Hagenwisch	do	4		do	do	Do.
15	Wm. W. McFarlane	Fingerboard road, Southfield, N. Y	2		do	do	Do.
15	John Johnson	Sand lane, Southfield, N. Y	2		do	do	Do.
15	John Scott	Vanderbilt avenue, Southfield, N. Y	6		do	do	Do.

18	Joseph Zurn	Concord street, Southfield, N. Y	4		do	do	Do.
18	Jas. McCree	do	4		do	do	Do.
18	Henry Frochell	do	4		do	do	Do.
18	Mr. Fisher	Fingerboard road, Southfield, N. Y	4		do	do	Do.
18	Lew Walters	do	5		do	do	Do.
18	L. H. Meyer	do	13		do	do	Do.
18	Mrs. Luling	do	5		do	do	Do.
18	C. Simonson	do	6		do	do	Do.
18	L. Kost	Clifton House, Southfield, N. Y	4		do	do	Do.
19	Camp Scott	R. W. Cameron street, Southfield, N. Y	4		do	do	Do.
19	Lewis De Comean	New York avenue, Southfield, N. Y	3	(*)	do	do	Do.
18	Fort Wadsworth	do	2		do	do	Do.
19	Mr. Haxton	South Beach, Southfield, N. Y	2		do	do	Do.
19	Geo. Ockerhausen	do	2		do	do	Do.
19	Geo. Whitford	do	13	(*)	do	do	Do.
19	John Kettletash	do	3		do	do	Do.
19	Geo. Schneider	Sand lane, Southfield, N. Y	4		do	do	Do.
19	Mr. Alexander	Richmond avenue, Southfield, N. Y	5		do	do	Do.
19	I. K. Martin	do	2		do	do	Do.
19	Charles L. Townsend	Dutch Farm, Southfield, N. Y	2		do	do	Do.
20	Mr. Bowen	do	2		do	do	Do.
20	Louis Walff	do	1		do	do	Do.
20	Mr. Altman	do	2		do	do	Do.
20	Henry Henley	do	4		do	do	Do.
20	Mrs. Onely	do	2		do	do	Do.
20	John Merther	do	4		Poor	Poor	Do.
20	Robert Hoolahan	Concord street, Southfield, N. Y	5		do	do	Do.
20	Christian Zebender	do	15		Normal	Normal	Do.
20	James Silney	do	2		do	do	Do.
20	Mrs. Megroe	do	8		Poor	Poor	Do.
20	J. Ipp	do	4		Normal	Normal	Do.
20	Mr. Neadsell	do	4		do	do	Do.
20	Gattlel Vernon	do	1		do	do	Do.
20	Mr. Whitmore	do	1		do	do	Do.
20	Robert Conrad	do	1		do	do	Do.
22	Herman Baltzer	Fingerboard road, Southfield, N. Y	6		do	do	Do.
22	Mr. Gunshot	Dutch Farm, Southfield, N. Y	1		do	do	Do.
22	Mr. Sidell	do	1		do	do	Do.
22	Squire Force	Concord street, Southfield, N. Y	2		do	do	Do.
22	Levi O'Connor	do	3		Poor	Poor	Do.
22	Patt. Smith	do	4		do	do	Do.
22	P. Connelly	do	4		do	do	Do.
22	Jas. Staltzie	do	1		Normal	Normal	Do.
22	Mrs. Magengast	do	2		do	do	Do.
22	F. Foley	Richmond road, Southfield, N. Y	18		Poor	Poor	Do.
22	Dan'l Wandell	do	3		Normal	Normal	Do.
22	Mrs. Branner	do	12		do	do	Do.
22	Mr. Krause	do	2		do	do	Do.
25	John Smith	Old Town, Southfield, N. Y	2		do	do	Do.
25	Mrs. Cleaveland	do	10		do	do	Do.

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*One lost.

Investigation of contagious pleuro-pneumonia—Inspections on Staten Island, New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.							
Aug. 25	Tunis Butler	Richmond road, Southfield, N. Y	4		Normal	Normal	William Rose.
25	Geo. Alter	do	36		do	do	Do.
25	Mr. Perine.	do	8		do	do	Do.
25	Joseph Egbert	Garretson Station, Southfield, N. Y	5		do	do	Do.
25	Mr. Mayer	do	5		do	do	Do.
25	Mrs. Seaver	Richmond road, Southfield, N. Y	10		do	do	Do.
26	John Barnes	do	8		do	do	Do.
26	Mr. Smith	do	9		do	do	Do.
26	Mr. Ackerly	Grant City street, Southfield, N. Y	7		do	do	Do.
26	Mr. Castell	do	3		do	do	Do.
26	Wm. Berlach	do	2		do	do	Do.
26	Mr. Alexander	Toadt Hill, Southfield, N. Y	4		do	do	Do.
26	Samuel Warm	do	4		do	do	Do.
26	Judge Benedict	Farven street, Middleton, N. Y	16		do	do	Do.
26	William Fountain	Barton Turn, Southfield, N. Y	15		do	do	Do.
27	Patt Smith	Toad Hill, Middleton, N. Y	12		Poor	Poor	Do.
27	Mr. Green	do	2		do	do	Do.
27	Mr. Hughes	do	3		Normal	Normal	Do.
27	Mr. James	do	7		do	do	Do,
27	Patt Collins	Richmond avenue, Middleton, N. Y	14		Poor	Poor	Do.
27	Mr. Ostrander	Richmond road, Middleton, N. Y	3		Normal	Normal	Do.
27	Rev. Dr. Vogel	Moravian Church, Middleton, , N. Y.	8		do	do	Do.
27	W. W. Galloway	Richmond road, Middleton, N. Y	13		do	do	Do.
27	J. G. Galloway	New Dorp street, Southfield, N. Y	37		do	do	Do.
28	Judge Clawson	do	10		do	do	Do.
28	Robert Woodson	do	6		do	do	Do.
28	Wm. H. Vanderbilt	do	11		do	do	Do.
28	Washington Dill	do	3		do	do	Do.
28	Mrs. H. Britton	do	2		do	do	Do.
28	St. Stephen's Home for Children	Richmond road, Middleton, N. Y	8		do	do	Do.
28	Alonzo Pitler	South Beach, Southfield, N. Y	4		do	do	Do.
28	John Burbank	do	4		do	do	Do.
28	Dr. E. Clark	Clader Grove, Sonthfield, N. Y	9		do	do	Do.
29	Moses Greenwalt	South Beach, Southfield, N. Y	15		Poor	Poor	Do.
29	Robert Jones	New Dorp street, Southfield, N. Y	5		Normal	Normal	Do.
29	Several owners	Toad Hill, Middleton, N. Y	22		do	do	Do.
29	James Curoy	Egbert avenue, Middleton, N. Y	4		Poor	Poor	Do.

	29	Mrs. McVey	do	8		do	Normal	Do.
	29	S. Marsh	do	3		Normal	do	Do.
	29	Mrs. Rosenbergh	do	2		Poor	do	Do.
	29	Mr. Gundecker	do	4		do	do	Do.
	29	Mrs. Finley	do	6		do	do	Do.
	29	Mr. Nugent	do	6		do	Poor	Do.
	29	Mr. Bailey	do	2		Normal	Normal	Do.
	29	Mr. E. Emmons	do	3		do	do	Do.
	29	H. DeRoach	do	5		do	do	Do.
Sept.	1	Mr. Conner	Black Horse lane, Middleton, N. Y	1		do	do	Do.
	1	J. Freich	do	3		do	do	Do.
	1	Mr. Morgan	do	3		do	do	Do.
	1	Mr. Mead	do	4	(*)			Do.
	1	F. Curry	do	2		Normal	Normal	Do.
	1	Mr. Sullivan	do	3		do	do	Do.
	1	Mrs. Holmes	do	3		do	do	Do.
	1	Mr. Campbell	do	2		do	do	Do.
	1	Mrs. Welsh	do	2		do	do	Do.
	1	Mr. Paff	Richmond street, Middleton, N. Y	3		do	do	Do.
	1	R. C. Moore	do	3		do	do	Do.
	1	Dr. Yocum	do	12		do	do	Do.
	2	Mr. Chadsworth	do	1	1	do	P. P. C	Do.
Sept.	2	Mr. Geldner	do	33		do	Normal	Do.
	2	Mr. Prior	do	2		do	do	Do.
	2	Several owners	Moravian road, Middleton, N. Y	23			do	Do.
	3	Gen. Swift	Clove road, Middleton, N. Y	3		Normal	do	Do.
	3	Chas. Gannon	do	12		do	do	Do.
	3	Mrs. Trepole	do	3		do	do	Do.
	3	C. Hanley	do	7		do	do	Do.
	3	Clarence Manning	do	3		do	do	Do.
	3	Mr. Clingman	do	5	†1			Do.
	3	Mr. Drake	do	3		do	Normal	Do.
	3	Jas. Cochran	Brock avenue, Middleton, N. Y	3		do	do	Do.
	3	W. Bradley	do	16		do	do	Do.
	5	Gus. Miller	Petticoat lane, Middleton, N. Y	22		do	do	Do.
	5	Margaret Bean	William Brock street, Middleton, N. Y	4		do	do	Do.
	5	Mr. Koch	do	92		Abnormal	Carbuncular erysipelas.	Do.
	8	Mr. Layton	Morning Star road, Northfield, N. Y	26		Normal	Normal	Do.
	8	Patty Malony	Morning Star road, Gun-factory, N. Y	22		do	do	Do.
	8	Mr. Cutler	William Brock street, Northfield, N. Y	2		do	do	Do.
	8	Mr. DePew	Butcherville, Northfield, N. Y	3		do	do	Do.
	8	Dr. Tyson	Decker Turn street, Northfield, N. Y	13		do	do	Do.
	9	Israel Freeland	Turnpike, Northfield, N. Y	5		do	do	Do.
	9	Darcy Brothers	William Brock street, Northfield, N. Y	63		do	do	Do.
	9	Munroe Eckstine	Munroe road, Middleton, N. Y	2		do	do	Do.
	9	Mr. Crosherdun	Manor road, Middleton, N. Y	5		do	do	Do.
	9	Mr. Schaefer	do	7		do	do	Do.
	11	Mr. Latterett	Richmond Hill, Westfield, N. Y	24		do	do	Do.
	11	Fred. White	Manor road, Middleton, N. Y	9		do	do	Do.

*One lost.

† Dead.

Investigation of contagious pleuro-pneumonia—Inspections on Staten Island, New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884. Sept. 11	Capt. Schneider	Manor road, Middleton, N. Y	7		Normal	Normal	William Rose.
11	Mr. McHudren	do	11		do	do	Do.
11	Mr. Wellbrook	do	20		do	do	Do.
11	Mr. Avery	do	5		do	do	Do.
12	Poorhouse farms	do	8		do	do	Do.
12	Mr. Sneed	do	5		do	do	Do.
12	Mr, Schmidt	do	4		do	do	Do.
12	J. Dempsey	do	3		do	do	Do.
12	F. Simonson	do	8		do	do	Do.
12	Mr. Ford	do	7		do	do	Do.
12	Jas. Fenley	do	7		do	do	Do.
12	M. Purr	do	10		do	do	Do.
12	Dr. Patton	do	3		do	do	Do.
12	Sersemar Bross	Egbert avenue, Middleton, N. Y	32		do	do	Do.
15	Widow Sersemar	Manor road, Middleton, N. Y	12		do	do	Do.
15	Mr. Fisher	Egbert avenue, Middleton, N. Y	7		do	do	Do.
15	Mr. Fisher	Court-house Station, Southfield, N. Y	7		do	do	Do.
15	J. A. Las Vane	Gifford's street, Southfield, N. Y	2		do	do	Do.
15	Mr. Moore	do	1		do	do	Do.
15	Mr. Colon	do	2		do	do	Do.
15	Mr. Metcalf	do	1		do	do	Do.
15	Mrs. Brown	do	2		do	do	Do.
15	Jas. Guion	do	11		do	do	Do.
15	Major Guion	do	3		do	do	Do.
15	Dick Fitzgerald	do	2		do	do	Do.
15	Pat. Collins	do	2		do	do	Do.
15	Mr. Morney	do	4		do	do	Do.
16	Mr. Mattison	do	13		do	do	Do.
16	Mrs. Crook	Eltingville, Southfield, N. Y	2		do	do	Do.
16	Mr. Hooper	do	1		do	do	Do.
16	J. Egbert	do	2		do	do	Do.
16	Mr. Bullman	do	2		do	do	Do.
16	Mr. Bedlam	do	2		do	do	Do.
16	Mr. Conley	do	7		do	do	Do.
16	Mr. Mushrow	do	5		do	do	Do.
16	Mr. Samuels	Gifford's street, Southfield, N. Y	2		do	do	Do.
16	Mrs. Brower	do	5		do	do	Do.
16	Mr. Robinson	do	8		do	do	Do.

16	Mr. Gilberg	do	1		do	do	Do.
16	Mrs. Tanner	do	1		do	do	Do.
16	Geo. Colon	do	4		do	do	Do.
16	L. Garretson	do	8		do	do	Do.
17	G. W. White	Fresh Hills, Westfield, N. Y	136		do	do	Do.
17	Mrs. E. Banker	do	3		do	do	Do.
17	Mr. Garrettson	do	3		do	do	Do.
17	C. Wallf	do	2		do	do	Do.
17	Mrs. McCoy	do	1		do	do	Do.
17	Mr. Goft	do	8		do	do	Do.
18	Mr. Butler	do	3		do	do	Do.
18	Aleck. Powell	do	3		do	do	Do.
18	J. Guion	do	3		do	do	Do.
18	Mrs. Gory	do	2		do	do	Do.
18	Mr. Hewitt	do	7		do	do	Do.
18	Mr. Madison	do	12		do	do	Do.
18	Mr. Patton	do	4		do	do	Do.
18	Mr. Rink	do	7		do	do	Do.
18	M. Concklin	Eltingville, Westfield, N. Y	6		do	do	Do.
18	Mr. Dunn	do	2		do	do	Do.
18	Mr. Dulan	do	2		do	do	Do.
18	Mr. Mishoo	do	1		do	do	Do.
18	Mrs. Geritey	do	3		do	do	Do.
19	Pat Boyle	do	6		do	do	Do.
19	Matt Rice	do	2		do	do	Do.
19	Mr. Horner	do	2		do	do	Do.
19	Nelson Gibley	Annadale street, Westfield, N. Y	2		do	do	Do.
19	Mr. Cortleon	do	2		do	do	Do.
19	Mr. Cole	do	2		do	do	Do.
19	Mr. Hunt	do	1		do	do	Do.
19	Mr. Roberts	do	4		do	do	Do.
19	Mr. Cooper	do	2		Poor	do	Do.
19	Mr. Gurnip	do	12		Normal	do	Do.
19	G. Burns	do	1		do	Poor	Do.
19	Robert Ennize	do	6		Poor	Normal	Do.
19	J. Fitzpatrick	do	4		do	do	Do.
22	S. Herman	do	1		Normal	do	Do.
32	Mr. Taylor	do	1		do	do	Do.
22	E. H. Jessup	do	5		do	do	Do.
22	Mr. Freeman	do	2		do	do	Do.
22	J. Tobin	Huguenot street, Westfield, N. Y	30		do	do	Do.
22	Boyle Bros. & Co	do	17		do	do	Do.
22	Mr. Parker	do	35		do	do	Do.
22	H. H. Benedict	do	6		do	do	Do.
22	Laurence Herr	do	3		do	do	Do.
22	Mr. Hessner	do	2		do	do	Do.
23	J. Laforge	Prince's Bay street, Westfield, N. Y	5		do	do	Do.
23	Henry Seguine	Prince's Bay, Westfield, N. Y	12		do	do	Do.
23	A. H. Johnson	do	85		do	do	Do.
24	Mr. Bedel	Pleasant Plains, Westfield, N. Y	2		do	do	Do.
24	L. G. Webster	do	2		do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections on Staten Island, New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.							
Sept. 24	Wm. Sharrot	Pleasant Plains, Westfield, N. Y	4		Normal	Normal	William Rose.
24	Mr. Seguine	do	12		do	do	Do.
24	Father Drumgoold	Richmond Valley, Westfield, N. Y	40		do	do	Do.
24	Eddy Kreischer	do	6		do	do	Do.
26	Henry Kreischer	Kreischerville, Westfield, N. Y	3		do	do	Do.
26	B. Kreischer	do	4		do	do	Do.
26	J. Androveth	do	2		do	do	Do.
26	Mr. Du Boise	do	4		do	do	Do.
26	Mr. Leak	do	7		do	do	Do.
26	G. Ellis	do	5		do	do	Do.
26	J. Hopping	Tottenville, Westfield, N. Y	1		do	do	Do.
26	G. Frazier	do	3		do	do	Do.
26	Wm. Drake	do	2		do	do	Do.
26	G. H. Haight	do	3		do	do	Do.
26	John Drake	do	13		do	do	Do.
26	E. Elliott	do	2		do	do	Do.
26	Mr. Sharrott	do	2		do	do	Do.
26	Mr. Brogan	do	5		do	do	Do.
27	Mr. McKinney	Rossville, Westfield, N. Y	3		do	do	Do.
27	Mr. Deynise	do	5		do	do	Do.
27	Mr. Wolff	do	2		do	do	Do.
27	H. Seguine	do	4		do	do	Do.
27	Mr. Nash	do	3		do	do	Do.
27	Peter McKune	do	4		do	do	Do.
27	Mr. Jessup	Rossville, Westfield, N. Y	5		do	do	Do.
27	Mr. Buckley	do	2		do	do	Do.
27	Mr. Dixon	do	1		do	do	Do.
27	Johnson Winnant	do	1		do	do	Do.
27	Joseph Perine	do	4		do	do	Do.
27	P. B. Laforge	Wood Row, Westfield, N. Y	4		do	do	Do
27	A. Eddy	do	3		do	do	Do.
27	H. Decker	do	8		do	do	Do.
27	Hiram B. Cropsey	do	4		do	do	Do.
27	J. W. Seguin	do	4		do	do	Do.
29	J. Callahan	New Brighton, Castleton, N. Y	17		do	do	Do.
29	Mr. Hare	do	2		do	do	Do.
29	Mrs. Trendle	New Brighton, Castleton, N. Y	5		do	do	Do.
29	Mrs. McNalley	do	6		do	do	Do.

	29	Mr. Carter	Castleton avenue, Castleton, N. Y	6		do	do	Do.
	29	Pat. Lynch	Davis avenue, Castleton, N. Y	25		do	do	Do.
	29	Mrs. Blane	Lafayette avenue, New Brighton, N. Y	7		do	do	Do.
Oct.	1	Richard Ahearn	Montgomery street, Castleton, N. Y	7		do	do	Do.
	1	J. Healey	do	8		do	do	Do.
	1	Mr. Davis	do	3		do	do	Do.
	1	McCarroll	Castleton avenue, New Brighton, N. Y	30		do	do	Do.
	1	John Mack	Tompkins avenue, New Brighton, N. Y	1		do	do	Do.
	1	Pat. McClennon	do	1		do	do	Do.
	1	Frederick Meissner	do	2		do	do	Do.
	1	Mrs. Caldwell	Montgomery avenue, New Brighton, N. Y	2		do	do	Do.
	1	Mrs. Hartley	do	1		do	do	Do.
	1	Mr. Herpeck	New Brighton, N. Y	5		do	do	Do.
	2	Erastus Wyman	Tompkins avenue, New Brighton, N. Y	2		do	do	Do.
	2	Mr. Wienor	Central avenue, New Brighton, N. Y	3		do	do	Do.
	2	Mr. Telfair	do	6		do	do	Do.
	2	Mr. Kribs	do	2		do	do	Do.
	2	Mr. Irwin	do	1		do	do	Do.
	2	Mr. Stone	Cotton Docks, New Brighton, N. Y	2		do	do	Do.
	2	Mrs. Green	Stokes avenue, New Brighton, N. Y	5		do	do	Do.
	2	Mr. Stokes	do	5		do	do	Do.
	2	Mr. Bogart	do	2		do	do	Do.
	2	Silas Havens	Jersey street, New Brighton, N. Y	14		do	do	Do.
	2	Henry Bath	do	2		do	do	Do.
	2	Mr. Brown	do	1		do	do	Do.
	2	Mr. Driscool	do	2		do	do	Do.
	2	Mr. Holtcamp	do	2		do	do	Do.
	3	Mr. Berts	do	3		do	do	Do.
	3	Mr. Read	Cedder Hill, New Brighton, N. Y	1		do	do	Do.
	3	Mr. Johnerty	New Brighton Terrace, New Brighton, N. Y	2		do	do	Do.
	3	M. L. Fowler	do	1		do	do	Do.
	3	Mr. McFaris	do	3		do	do	Do.
	3	Mr. Radewald	Clinton avenue, New Brighton, N. Y	2		do	do	Do.
	3	Mr. McCartey	do	2		do	do	Do.
	3	J. R. Rusk	do	3		do	do	Do.
	3	Mr. Bailey	Bard avenue, New Brighton, N. Y	1		do	do	Do.
	3	Geo. W. Curtis	do	1		do	do	Do.
	3	Major Delafield	do	2		do	do	Do.
	3	R. B. Minturn	do	3		do	do	Do.
	3	Mr. Carey	do	2		do	do	Do.
	3	Dr. S. Elliott	do	2		do	do	Do.
	3	Mr. Taylor	do	4		do	do	Do.
	3	Mr. Shaw	Davis avenue, New Brighton, N. Y	3		do	do	Do.
	6	Luke Ward	Castleton avenue, New Brighton, N. Y	35		do	do	Do.
	6	Trustees Disabled Sailors	Sailors' Snug Harbor, Brighton, N. Y	13		do	do	Do.
	6	Mr. Davis	Bard avenue, New Brighton, N. Y	3		do	do	Do.
	6	L. H. Hoyt	do	3		do	do	Do.
	7	Mike Conelly	Brighton avenue, West Brighton, N. Y	3		do	do	Do.
	7	F. Emmons	Burgher avenue, New Brighton, N. Y	3		do	do	Do.
	7	Mrs. Lions	do	5		do	do	Do.
	7	Mr. Carroll	do	1		do	do	Do.
	7	Pat. Fitgerald	do	1		do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections on Staten Island, New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.							
Oct. 7	Mr. Whiteing	Beaumont avenue, West Brighton, N. Y	1		Normal	Normal	William Rose.
7	Mr. Suter	Pelton Place, West Brighton, N. Y	5		do	do	Do.
7	Mr. Woods	Franklin avenue, West Brighton, N. Y	2		do	do	Do.
7	Mr. Donovan	do	3		do	do	Do.
7	Mrs. Dugan	do	12		Poor	Poor	Do.
7	Mr. Roach	Henderson avenue, West Brighton, N. Y	1		Normal	Normal	Do.
7	Stewart Brown	do	6		do	do	Do.
7	Mrs. Johnson	do	3		do	do	Do.
7	Mrs. McAlvey	do	6		do	do	Do.
7	Mr. Hagerty	do	2		do	do	Do.
7	Mrs. Dunaring	do	3		do	do	Do.
7	Mr. McQuade	Broadway avenue, West Brighton, N. Y	6		do	do	Do.
7	Mr. Calbert	do	2		do	do	Do.
7	David Campbell	do	3		do	do	Do.
7	Mrs. Bedor	Taylor street, West Brighton, N. Y	1		do	do	Do.
7	Mr. McCarthey	do	5		do	do	Po.
7	Mr. Parker	do	3		do	do	Do.
7	Mr. Craton	do	2		do	do	Do.
9	Mr. Bodine	Mill road, West Brighton, N. Y	2		do	do	Do.
9	Mr. Mayer	do	12		Poor	do	Do.
9	Mr. King	Broadway, West Brighton, N. Y	3		Normal	do	Do.
10	Mrs. N. Barrett	do	3		do	do	Do.
10	Col. F. C. Barrett	Castleton Heights, West Brighton, N. Y	2		do	do	Do.
10	Maj. C. Barrett	do	3		do	do	Do.
10	Mr. H. Britton	Clove road, Castleton, N. Y	6		do	do	Do.
10	Mrs. Britton	do	2		do	do	Do.
10	Mr. Barns	do	6		do	do	Do.
10	Mr. Gibson	do	4		do	do	Do.
10	Mr. Carpenter	Columbia street, Castleton, N. Y	1		do	do	Do.
10	Mrs. Degerest	do	2		do	do	Do.
10	Covey & Brother	do	6		do	do	Do.
10	Mrs. McCue	do	2		do	do	Do.
10	Mrs. Lynch	do	2		do	do	Do.
10	Mr. McCalt	do	5		do	do	Do.
10	Erastus Brooks	do	8		do	do	Do.
13	Mr. Martling	Manor road, Northfield, N. Y	3		do	do	Do.
13	Mr. Raymond	do	4		do	do	Do.
13	Captain Smith	do	8		do	do	Do.

13	Mrs. Jerritt	Jude avenue, Northfield, N. Y	8		do	do	Do.
13	Mr. James	Shore road, Northfield, N. Y	5		do	do	Do.
13	Henry Bealaugh	do	3		do	do	Do.
13	Mr. Matthiasson	Church road, Northfield, N. Y	25		do	do	Do.
13	Mr. Redman	Richmond road, Northfield, N. Y	25		do	do	Do.
14	Mr. Reupen	Grantville, New York	12		do	do	Do.
14	Mrs Krone	Grantville, Northfield, N. Y	2		do	do	Do.
14	A. Winnant	do	4		do	do	Do.
14	Mr. Jeffrey	do	30		do	do	Do.
14	Mr. Decker	Butcherville, Northfield, N. Y	14		do	do	Do.
14	Mr. Egbert	do	16		do	do	Do.
14	Mr. Pew	do	3		do	do	Do.
14	Mr. Christopher	Springfield, Northfield, N. Y	8		do	do	Do.
16	All. Vrame	do	5		do	do	Do.
16	Mrs Shiloah	Springville, Northfield, N. Y	13		do	do	Do.
16	Mr. Trifkee	do	8		do	do	Do.
16	Mr. Molerk	do	8		do	do	Do.
16	Mr. Stabler	Bull's Head, Northfield, N. Y	4		do	do	Do.
16	Mr. Burnham	do	2		do	do	Do.
16	Mr. Simonson	do	1		do	do	Do.
16	J. Christopher	do	1		do	do	Do.
16	Mr. Gardner	do	9		do	do	Do.
16	Moses Maybaum	Linoleumville, Northfield, N. Y	5		do	do	Do.
17	Pat. McKune	do	4		do	do	Do.
17	Mr. Denyse.	do	5		do	do	Do.
17	Mrs. Seguine	do	8		do	do	Do.
17	Mr. Nash	do	2		do	do	Do.
17	Mr. Wohlfer	Traversville, Northfield, N. Y	2		do	do	Do.
17	Mr. Jesup	do	5		do	do	Do.
17	Mr. Buckley	do	2		do	do	Do.
17	Mr. Morris	Redmond turnpike, Northfield, N. Y	15		do	do	Do.
20	Mr. Crosherm	Grantville, Northfield, N. Y	9		do	do	Do.
20	J. Winant	do	8		do	do	Do.
20	Mrs. Gurnoy	do	2		do	do	Do.
20	Mr. Day	do	2		do	do	Do.
20	Mr. Ecker	do	8		do	do	Do.
20	Mr. Tynam	Richmond turnpike, Northfield, N. Y	1		do	do	Do.
20	Mr. Link	do	2		do	do	Do.
20	Mr Davidson	do	2		do	do	Do.
20	Jas. Hunt	do	8		do	do	Do.
20	Mr. Mushrow	do	2		do	do	Do.
20	Mr. Sage	do	1		do	do	Do.
21	Mr. Otis	do	1		do	do	Do.
21	Mr. Hyleman	Morning Star road, Northfield, N. Y	8		do	do	Do.
21	Abe Deckar	do	2		do	do	Do.
21	Mr. Mushrow	do	2		do	do	Do.
21	Mr. Thompson	Northfield road, Northfield, N. Y	2		do	do	Do.
21	Mr. Maloy	Morning Star road, Northfield, N. Y	26		do	do	Do.
21	Mr. Vreeland	do	2		do	do	Do.
21	Mr. Tyson	do	2		do	do	Do.
21	Mr. Hornby	do	10		do	do	Do.
21	Mr. Trew	Willbrook lane, Northfield, N. Y	6		do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections on Staten Island, New York—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.							
Oct. 21	Mr. Ensinger	Willbrook lane, Northfield, N. Y	5		Normal	Normal	William Rose.
22	Gus. Miller	Bull's Head, Northfield, N. Y	15		do	do	Do.
22	J. Lake	do	4		do	do	Do.
22	Mr. Dray	do	3		do	do	Do.
22	Mr. Cutter	do	8		do	do	Do.
22	Mrs. Blaine	do	85		do	do	Do.
22	S. Stanling	do	1		do	do	Do.
22	Mr. Derome	do	2		do	do	Do.
23	Mr. Hughes	do	2		do	do	Do.
23	Mr. Blake	do	2		do	do	Do.
23	Mr. Hauseman	do	1		do	do	Do.
23	Mr. Martin	do	1		do	do	Do.
23	Mr. Clevo	do	1		do	do	Do.
23	Mr. Vrome	do	5		do	do	Do.
23	Mr. Platts	do	3		do	do	Do.
24	Mr. Kissing	Summerville, Northfield, N. Y	18		do	do	Do.
24	Mr. Merrick	Himerville, Northfield, N. Y	16		do	do	Do.
24	Mr. Redman	do	25		do	do	Do.
24	Mr. Merrill	do	9		do	do	Do.
24	Mr. Agan	do	1		do	do	Do.
24	Mr. Hunt	do	12		do	do	Do.
24	Mr. Dunkee	do	7		do	do	Do.
24	Mr. Selig	do	20		do	do	Do.
27	Mr. Latcorett	Richmond Hill, Middleton, N. Y	11		do	do	Do.
27	Mr. Sholes	Chelsea, Northfield, N. Y	8		do	do	Do.
27	Mrs. Morleck	do	3		do	do	Do.
27	Mr. Simonson	do	2		do	do	Do.
27	Mr. Merrill	do	8		do	do	Do.
27	Mr. Blum	do	7		do	do	Do.
27	J. Dunkee	do	2		do	do	Do.
27	Mr. Dunkee	do	4		do	do	Do.
27	Mrs. Perkins	Richmond turnpike, N. Y	4	1	Poor	Poor	Do.
27	Mrs. Lynch	Stapleton, Northfield, N. Y	3		Normal	Normal	Do.
28	The Commons	Campscott, Southfield, N. Y	306		do	do	Do.
28	Mr. Freeman	Richmond road, Middleton, N. Y	17		do	do	Do.

INSPECTIONS IN NEW JERSEY.

June 28	John Hall estate	Farm of Ounri, New Jersey	55	3			W. B. E. Miller.
July 29	Elmri Goldy	Main street, Hightstown	1				Do.
29	John V. Ely	Public road, Milford	20	2	Excellent	Good	Do.
29	John Norton	do	18		Good	do	Do.
29	Wm. C. Hutchinson	Walker avenue, Allentown	1	1			Do.
Aug. 4	John Norton	Public road, Milford	18		Good	Good	Do.
4	John V. Ely	do	20	2	do	do	Do.
11	Messrs. Bettle & Ebans	White House, Camden	37	2	do	do	Do.
Oct. 18	Wm. S. Taylor	Jacksonville, Burlington	45		do	do	Do.
Nov. 3	Henry Wetmer	Race-Course road, Secaucus	1			do	Rowland & Hawk.
3	Henry Liffield	Secaucus	9			do	Do.
3	M. Johnson	do	4			do	Do.
3	Mrs. Engletree	do	2			do	Do.
3	A. Litherman	do	60		Filthy	Not good	Do.
3	M. R. Churchhill	do	3			do	Do.
3	F. Wunders	do	3			do	Do.
3	C. H. Hops	do	25			do	Do.
3	W. H. Huber	do	4			do	Do.
4	Mr. J. C. Vogs	do	27	2		Fair	Do.
4	Hyman Fisher	do	9	1		do	Do.
4	A. Gutjahr	do	9	1			Do.
4	Geo. Hichlich	do	2			Good	Do.
4	J. Block	do	27	2		Fair	Do.
4	Mr. Brockwell	do	9	1		Not good	Do.
4	S. Koslowsky	do	16	3		Fair	Do.
4	Geo. Harmes	do	9	1			Do.
1	Mr. A. Kellar	Ridgewood	27			Good	Do.
1	Mr. Sing	do					Do.
1	Joseph Lang	Williams street, Ridgewood	14	1		Fair	Do.
1	Henry Doht	Wickoff avenue, Ridgewood	53	6			Do.
1	Philip Reid	Ridgewood	25			Good	Do.
3	A. Cucher	Secaucus	5			do	Do.
3	George Hicklick	Secaucus avenue, Secaucus	2			do	Do.
4	Thomas Dwyer	Secaucus	1			do	Do.
4	Jacob Bolary	do	3			do	Do.
5	John Kirkpatrick	do	3			do	Do.
5	Fred. Kasper	do	9			do	Do.
5	H. Smooger	do	1			do	Do.
5	John Kuntz	do	12			do	Do.
5	H. Helmke	do	25			do	Do.
5	George Flegenstine	Near Secaucus	14	1		Fair	Do.
6	Geo. Breumst	Hague street, Secaucus	11			Good	Do.
6	Thomas Islim	Near Secaucus	5			do	Do.
6	John Farling	do	10			do	Do.
6	N. Horgan	do	13			do	Do.
6	Fred. Eythe	do	3			do	Do.
6	Patrick Coyle	do	9			do	Do.
6	Mrs. Taye	do	3			do	Do.

Investigation of contagious pleuro-pneumonia—Inspections in New Jersey—Continued.

Date.		Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.								
Nov.	7	Jersey City Abbattoir	Jersey City				Good	Rowland & Hawk.
	8	Jerry Conklin	Secaucus	7			do	Do.
	8	B McMahon	West Hoboken	7	1		Not good	Do.
	8	Mrs. Hand	Secaucus	5			Good	Do.
	8	Mrs. Kates	West Hoboken	11			do	Do.
	8	Wm. Smeeders	do	10			do	Do.
	8	M. Stockfish	do	26			do	Do.
	8	David Van Vorse	do	30			do	Do.
	8	Mrs. Jacob Swartz	do	1			Good	Do.
	8	Geo. Katlin	do	8			do	Do.
	8	Thomas Phoser	do	5			do	Do.
	8	John Engle	do	10			do	Do.
	8	W. Westerfelt	do	4			do	Do.
	8	Wintner Beck	do	5			do	Do.
	8	Patrick Mally	do	10			do	Do.
	8	Walter Alexander	do	15			do	Do.
	10	Fred. Vonberon	do	6			do	Do.
	10	M. Van Vosse	do	8			do	Do.
	10	Mr. Jean Claude	do	5			do	Do.
	10	Patrick Brady	do	15			do	Do.
	10	N. Fenning	do	7			Fair	Do.
	10	Mrs. Ormsbey	do	2			Good	Do.
	10	Mrs. Mooney	do	7			do	Do.
	10	Mrs. Plogg	do	1			do	Do.
	10	Charles Theele	do	1			do	Do.
May	5	Mr. Shocraft	Desbrosses street, Jersey City Heights	1				Do.
	6	Mr. Nolan	do	4				Do.
	14	Mr. Katz	Pavonia Ferry, Greenville	1				Do.
			Desbrosses street, Jersey City Heights	2				Do.
			do	4				Do.
			Pavonia Ferry, Jersey City	1				Do.
Nov.	11	John Lumis	Deckertown, Sussex	65		Very fine	Very fine	Do.
	11	J. W. Quick	do	13			Good	Do.
	11	W. E. Caskey	do	10			do	Do.
	11	Sam. R. Lawson	do	28			do	Do.
	11	B. J. Swartz	do	18			do	Do.
	11	John Kirldoff	do	18			do	Do.
	11	William Stiles	do	20			do	Do.
	12	Wm. Titsworth	do	25			do	Do.

12	J. L. Dewitt	Libertyville, Sussex	28			do	Do.
12	John Decker	do	18				Do.
12	M. Wright	Quarryville, Sussex	28			Good	Do.
12	John Casty	do	14			do	Do.
12	J. L. Quick	do	24			do	Do.
12	John Wilson	Fulton street, Weehawken	3	2		Not good	Do.
14	Thee. Hoberman	West Hoboken	17			do	Do.
14	Adams Altig	do	16			do	Do.
14	Peter Drickle	do	3			do	Do.
14	F. Voke	do	10			do	Do.
14	John Shatford	do	18			do	Do.
14	John Freeman	Union Hill	5			do	Do.
14	John Garner	do	6			do	Do.
14	B. Feeney	do	2			do	Do.
14	M. Carkers	do	10			do	Do.
14	Julius Vanerty	do	8			do	Do.
14	M. Shaw	do	6			do	Do.
14	M. Olmires	do	29			do	Do.
14	M. Fud	do	23			do	Do.
17	J. I. Wysinger	Little Falls	2			Fair	Do.
17	Fred Van Ayers	Road to Two Bridges, Little Falls	5			Good	Do.
17	H. Ryer	Little Falls	4			do	Do.
17	H. W. Hillstraw	do	2			do	Do.
17	H. Stanley	do	8			do	Do.
17	James Stevens	do	18			do	Do.
17	W. Kiser	do	25		Good	do	Do.
17	D. Covenhover	do	1		do	do	Do.
17	M. Habey	do	2		do	do	Do.
17	M. Petenger	do	4		do	do	Do.
17	John Candfield	do	32		do	do	Do.
17	Julius Finslip	Near Mount Clair	18		do	do	Do.
20	Henry Knapp	Newark	9		do	do	Do.
20	Augustus Caufault	do	26		do	do	Do.
20	Lewis Webert	do	11		do	do	Do.
20	Anton Beeler	do	16		do	do	Do.
20	Wm. Sling	do	34		do	do	Do.
20	J. L. Wilson	do	9		do	do	Do.
20	Edward Franklin	Lester street, Newark	31		do	do	Do.
21	Annie Styles	Near Waverly	10		do	do	Do.
21	Andie Shriver	do	12		do	do	Do.
21	Isodore Himpley	do	10		do	do	Do.
21	James Mulliger	do	18		do	do	Do.
21	Wm. Pelferton	do	21		do	do	Do.
21	Henry Heeb	do	7		do	do	Do.
21	C. Kossel	do	25		do	do	Do.
21	Henry Myers	do	12		do	do	Do.
21	Mrs. J. E. Priest	Meek lane, near Waverly	12		do	do	Do.
21	Mike Swartz	Near Waverly	8		do	do	Do.
22	Henry McCandler	Near Linder	18		do	do	Do.
22	Thomas McCandler	do	19		do	do	Do.
22	Geo. McGillery	do	10		do	do	Do.
22	Morse Bro	do	26		do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections in New Jersey—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.							
Nov. 22	Geo. G. Handcock	Near Linder	10		Good	Good	Rowland & Hawk.
22	Moses Winans	do	26		do	do	Do.
22	Clarence Winans	do	11		do	do	Do.
22	George W. Baker	do	7		do	do	Do.
22	W. A. Baker	do	9		do	Fair	Do.
22	Job Winans	do	16		do	Good	Do.
24	F. Blank	do	95		do	do	Do.
24	Urual Tucker	do	10		do	do	Do.
24	Donald Ross	do	12		do	do	Do.
24	Wm. Carmady	do	40		do	do	Do.
24	John Gragby	Rahway avenue, Linder	9		do	do	Do.
24	C. Kane	Morris avenue, Linder	12		do	do	Do.
24	A. Van Fleet	do	11		do	do	Do.
24	G. Hipping	do	18		do	do	Do.
24	John Duvemous	Lion's Farm	10		do	do	Do.
1	Evans & Beetle	White Horse Road, Camden	35		Good	do	W. B. E. Miller.
1	Joseph Collings	do	28		do	do	Do.
1	Joseph Hollingshead	do	26		do	do	Do.
3	A. D. Newall	Road to Washington, New Brunswick	19		do	do	Do.
3	Ellis Faimer	Road to Cranberry, New Brunswick	11		do	do	Do.
3	Jesse Jimeson	do	3		Fair	Fair	Do.
4	Hugh Sutherland	Bergen plank road, Jersey City	8		Good	Good	Do.
4	Bartlett Moore	do	3		do	do	Do.
4	James Cunningham	do	10		do	do	Do.
4	Joseph Neville	do	2		do	do	Do.
4	Samuel Kilpatrick	do	2		do	do	Do.
5	Timothy Murphy	200 Ocean avenue, Jersey City	1		do	do	Do.
5	Michael Cavanaugh	215 Ocean avenue, Jersey City	1		do	do	Do.
5	Stephen Conelly	407 Ocean avenue, Jersey City	7		do	do	Do.
5	John Estell	Ocean avenue, Jersey City	31	2	do	do	Do.
6	Chetwood Estell	17 Kearney avenue, Jersey City	13		do	do	Do.
6	Wm. Shaw	Near Bergen avenue, Jersey City	15		do	do	Do.
6	Alexander McNab	do	1		do	do	Do.
6	Wm. Wilson	Boswick avenue, Jersey City	1		do	do	Do.
6	Charles D. Whitall	12 Boswick avenue, Jersey City	1		do	do	Do.
6	Ira Browne	Bergen avenue, Jersey City	14		do	do	Do.
8	John V. Ely	Public road, Millford	19		do	do	Do.
10	Thomas Farrington	Kearney avenue, Jersey City	1		do	do	Do.
10	Joseph Hurley	45 Kearney avenue, Jersey City	3		do	do	Do.

	10	John Ertle	Kearney avenue, Jersey City	7		do	do	Do.
	10	Owen Shannon	13 Virginia avenue, Jersey City	2	1	do	do	Do.
	12	John Hall (estate)	Near Freehold	49		do	do	Do.
	13	Mrs. Rush	50 Edgar avenue, Jersey City	3	2	Fair	Fair	Do.
	13	Mrs. Beigman	42 Edgar avenue, Jersey City	8	1	Good	Good	Do.
	13	John Foster	119 Edgar avenue, Jersey City	7		do	do	Do.
	13	R. G. Vruland	Ege avenue, Jersey City	8		do	do	Do.
	13	C. Soltau	11 Ege avenue, Jersey City	1		do	do	Do.
	13	Frederick Harm	Road to Jobstown, Columbus	30		do	do	Do.
	17	George Roach	West Bergen avenue, Jersey City	25		do	do	Do.
	17	Wm. Koelsch	West side avenue, Jersey City	3		do	do	Do.
	17	E. A. Rapp	do	1		do	do	Do.
	17	Wm. A. Kimball	Culver avenue, Jersey City	1		do	do	Do.
	17	Luther Cummings	West Bergen avenue, Jersey City	1		do	do	Do.
	17	E. Cole	do	1		do	do	Do.
	17	W. H. Chattock	do	1		do	do	Do.
	20	Major Moore	Road to Jersey City, Rutherford	16		do	do	Do.
	21	William Walton	Road to Princeton, Dutch Neck	28		do	do	Do.
	24	Theodore Yager	Grand avenue, Jersey City	4		do	do	Do.
	24	James Nordham	Ege avenue, Jersey City	4		do	do	Do.
	24	Mrs. Blank	do	7		Excellent	Excellent	Do.
	24	Edward McKeon	Grand avenue, Jersey City	2		do	do	Do.
	24	Wm. Kavenaugh	Ege avenue, Jersey City	1		Good	Good	Do.
	26	J. R. Bucke	Ocean avenue, Jersey City	11		do	do	Do.
	26	Nathan Robinson	do	4		do	do	Do.
	26	Owen Ramsby	do	10		do	do	Do.
	26	A. R. & G. S. Mitchell	Gove street, Orange	91		Very fine	Very fine	Rowland & Hawk.
	26	M. M. Dodd	do	47		do	do	Do.
	26	Booth & Dorer	do	45		Good	Good	Do.
	26	H. W. Gilbertson	do	38		do	do	Do.
	28	T. H. Pierson	Arlington avenue, Orange	27		do	do	Do.
	28	Febie A. Pierson	do	20		do	do	Do.
	28	N. Nall	Arlington avenue, East Orange	34		do	do	Do.
	28	F. H. Baldwin	Walnut street, East Orange	16		do	do	Do.
	28	Conrad Nolty	Gove street, East Orange	34		do	do	Do.
	28	K. Cissner	South Orange	8		do	do	Do.
	28	Valor Dersinger	do	8		do	do	Do.
	28	F. Newhouse	do	27		do	do	Do.
	29	Geo. Harms	Bevistel street, Secaucus	9	1	Fair	Fair	Do.
	29	Frank Killer	do	5	1	do	do	Do.
	29	S. Koslowsky	do	15		Not good	Not good	Do.
Dec.	2	John Wilson	Fulton street, Weehawken	3				Do.
	4	Sam. Greenleaf	Hackensack road, Durham	10	4	Not good	Poor	Do.
Nov.	22	Taylor's Stock Yards	Trenton, New Jersey	42				W. B. E. Miller.
Dec.	1	James Rodgers	East Newark	8			Good	Rowland & Hawk.
	1	Jacob Maurr	7th street, East Newark	7			do	Do.
	1	James Braslin	East Newark	5			do	Do.
	1	Edward Fitzgerald	do	4	1	Fair	Fair	Do.
	1	P. Lion	Plank road, East Newark	11				Do.
	1	Thom. Kellet	East Newark	8			Good	Do.
	1	Andrew J. Miller	do	7			do	Do.
	1	John Martin	Foundry street, East Newark	16			do	Do.

Investigation of contagious pleuro-pneumonia—Inspections in New Jersey—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.							
Dec. 1	Edward Ryan	Plank road, East Newark	3			Good	Rowland & Hawk.
1	Henry Payfer	Warren street, East Newark	17			do	Do.
1	James Peck	East Orange	6			do	Do.
1	Richard White	Park street, East Orange	13			do	Do.
1	John Dorsal	East Orange	7	1		Fair	Do.
1	Wm. Nichols	West street, East Orange	6			Good	Do.
2	H. Frick	East Orange	18			do	Do.
2	P. S. Hayward	do	11			do	Do.
2	Wm. H. Fredericks	Clinton street, East Orange	20			do	Do.
2	S. O. Donald	do	9			do	Do.
2	Wm. Goerdes	Vailesburg	14			do	Do.
2	Catholic Institute	South Orange	14			do	Do.
2	Michael Dorah	do	12			do	Do.
2	Hermau Schench	do	10			do	Do.
2	P. F. Howell	do	24			do	Do.
2	John Herohouse	do	14			do	Do.
3	Wm. Hess	Bergen street, South Orange	2			do	Do.
3	Ezra Reeves	Irvington	13			do	Do.
3	Jacob Hess	do	10			do	Do.
3	John Ballard	do	24			do	Do.
3	John Shaffer	do	10			do	Do.
3	P. Bippard	Clinton street, Irvington	14			do	Do.
3	Elize Nitmen	Irvington	13				Do.
3	Andrew Burns	do	15				Do.
3	Mrs. M. Hall	do	6				Do.
3	C. W. Harrison	do	14			Good	Do.
3	F. Mutler	do	17			do	Do.
3	Charley Hodley	do	20			do	Do.
3	I. A. Oakley	do	17			do	Do.
3	M. Stickman	South Orange	6			do	Do.
3	Jas. Gribbs	do	7			do	Do.
4	Wm. Woodruff	do	21			do	Do.
4	Dan. Hadden	do	10			do	Do.
4	Fred. Hess	Wall street, South Orange	11			do	Do.
4	Mrs. M. Edwards	South Orange	7			do	Do.
4	Nicholas Olman	do	10			do	Do.
4	John Eizar	West Newark	20			do	Do.
4	Wm. Gogle	do	23			do	Do.
4	John Winter	do	5			do	Do.

5	Geo. W. Gan	Irvington	18			do	Do.
5	O. P. Littlefield	Springfield avenue, Irvington	28			do	Do.
5	Tincher Munn	Irvington	24			do	Do.
5	Patrick Larney	do	16			do	Do.
5	S. Carter	do	5			do	Do.
5	Westley Baldwin	do	25			do	Do.
5	Thomas Fitzpatrick	do	36			do	Do.
6	E. B. Jagers	Lion's Farm	12			do	Do.
6	James Chapman	do	10			do	Do.
6	Ralph Humphry	do	8			do	Do.
6	John Dempsey	do	17			do	Do.
6	F. Utess	Irvington	13			Good	Do.
6	K. Jeffries	do	12			do	Do.
6	John Hess	Elizabeth avenue, Irvington	14			do	Do.
6	Ferdnand Fegro	Bergen street, Irvington	11			do	Do.
6	T. B. Andrus	Clinton avenue, Irvington	10			do	W. B. E. Miller.
1	Anson Owens	Garfield avenue, Jersey City	17			do	Do.
1	John Nevins	do	18		Fair	Fair	Do.
1	James Messenger	do	6		Good	Good	Do.
1	Wm. Bays	do	6				Do.
1	Jas. Atchinson	Corner Clark and Clairmont, Jersey City	9		Good	Fair	Do.
1	Mr. Martin	23 Atlantic street, Jersey City	1	1			Do.
3	Wm. Burke	26 Atlantic street, Jersey City	13		Fair	Fair	Do.
8	Mrs. J. Growsche	Turhner street, Jersey City	8		do	do	Do.
3	Mrs. Thos. Hurley	Near Bergen avenue, Jersey City	8				Do.
3	Charles Ackley	117 Kearney street, Jersey City	6		Good	Good	Do.
3	John Ackley	130 Kearney street, Jersey City	5		Filthy	do	Do.
3	Frank Fanington	120 Kearney street, Jersey City	5	1	Fair		Do.
4	Mrs. Cabel	137 Fulton avenue, Jersey City	8		do	Fair	Do.
4	Frank Weisman	Fulton avenue, Jersey City	12		do	do	Do.
4	Mrs. Carson	145 Fulton avenue, Jersey City	2		Good	Good	Do.
4	Edward Shinn	60 Fulton avenue, Jersey City	2		do	do	Do.
4	Patrick Kelly	Near Walker avenue, Jersey City	4		do	do	Do.
4	John Murray	Rose street and Fulton avenue, Jersey City	13		Fair	Fair	Do.
5	Frank Palmer	Rose street, near Fulton avenue, Jersey City	10		Good	Good	Do.
5	John Palmer	do	11		do	do	Do.
5	R. Haroe	1082 Bergen street, Jersey City	13		do	do	Do.
5	Patrick Mulcahy	Bergen street, Jersey City	26		do	do	Do.
5	John Hannon	do	7				Do.
5	Frank Wooleon	Pamrapo avenue, Jersey City	19		Good	Good	Rowland & Hawk.
6	Jacob Kepler	124 Jackson lane, Jersey City	13		Fair	Fair	W. B. E. Miller.
6	Peter Wisner	Corner Jackson and Wight streets, Jersey City	2		Good	Good	Do.
6	Stephen Casey	49 Walker avenue, Jersey City	7		Fair	Fair	Do.
6	Luke Conneff	57 Walker avenue, Jersey City	9		do	do	Do.
6	Bernard O'Neil	213 Brittain street, Jersey City	10	5	Very filthy	do	Do.
7	Thomas Hearthley	81 Germania avenue, Jersey City	1		do	Good	Do.
7	Bernard Riley	70 Germania avenue, Jersey City	1		Good	do	Do.
7	George Reed	87 Germania avenue, Jersey City	5	1	do	do	Do.
7	Patrick Coyle	175 Bevins street, Jersey City	19	1	do	do	Do.
7	John Connor	432 Tunnelly avenue, Jersey City	2		Fair	Fair	Do.
9	James Madden	Saint Paul avenue, Jersey City	6	1	do	do	Do.
9	John Willhaven	do	13		do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections in New Jersey—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stables.	Condition of animals.	Name of inspector.
1884.							
Dec. 9	Benjamin Hill	97 Larch street, Jersey City	10		Clean	Good	W. B. E. Miller.
10	D. Monenbrock	26 Wales avenue, Jersey City	1		Fair	Fair	Do.
10	Martin Shugroff	43 Wales avenue, Jersey City	19	4	do	do	Do.
10	Fredericka Anletz	7 Pavonia avenue, Jersey City	17	5	do	do	Do.
10	John Reader	Wallace avenue, Jersey City	1		do	do	Do.
10	Lawrence Cullen	530 West Side avenue, Jersey City	18	3	Clean	do	Do.
10	John Foster	97 Stuyvesant avenue, Jersey City	2		Very filthy	Very bad	Do.
10	John Cullan	894 Montgomery street, Jersey City	10	2	Clean	Fair	Do.
10	August Smith	899 Montgomery street, Jersey City	8		do	Good	Do.
10	Lewis Stevins	West Side avenue and Duncan street, Jersey City	6		do	do	Do.
10	Mrs. Ellen Gillis	215 Olive avenue, Jersey City	4		Fair	Fair	Do.
10	Henry Poolsil	Belmont avenue, Jersey City	2		do	do	Do.
10	M. Dunn	190 Jeurth avenue, Jersey City	13		do	do	Do.
10	Mrs. Clark	Jeurth avenue, Jersey City	1		Good	Good	Do.
10	John Jackson	202 Jeurth avenue, Jersey City	8		do	Fair	Do.
10	Martin Connelly	Plainfield avenue, Jersey City	20		Filthy	Good	Do.
10	John Macey	do	1		Good	do	Do.
11	Owen Donahne	775 Communipaw avenue, Jersey City	4		do	do	Do.
11	Patrick Varney	85 Oxford street, Jersey City	1		do	do	Do.
11	John O'Keef	Glendening avenue, Jersey City	1		do	do	Do.
11	A. G. Welsh	90 Melville avenue, Jersey City	12	1	do	do	Do.
11	James Mrity	189 Glendening avenue, Jersey City	8		do	do	Do.
11	John Collins	112 Glendening avenue, Jersey City	5	1	Fair	Fair	Do.
11	John Lynch	Williams avenue, Jersey City	21	5	Filthy	do	Do.
11	Wm. Blacker	69 Hoadey avenue, Jersey City	10		Very good	Very good	Do.
11	Thomas Carey	Virginia avenue, Jersey City	6		do	Good	Do.
11	J. Bentze	200 Virginia avenue, Jersey City	3	1	do	do	Do.
11	Miss Vannable	Hatch & Venango avenue, Jersey City	1		do	do	Do.
8	P. Ballantine & Son	Near Waverly	9			do	Rowland & Hawk.
8	P. Lamence	do	9			do	Do.
8	M. C. Edgar	Lion's Farm	15			do	Do.
8	Wm. Marcell	do	39			do	Do.
8	W. H. Goldsmith	Clinton Place	8			do	Do.
8	Geo. Bowers	Irvington	16			do	Do.
8	E. Moasle	do	23			do	Do.
8	E. H. Underhill	do	29			do	Do.
8	P. F. Sommon	Waverly	2			do	Do.
8	Parker & Son	Irvington	14			do	Do.
8	C. Chamberlain	Waverly	23			do	Do.

8	James Platt	Irvington	8			do	Do.
11	Fred. Underhill	do	19			do	Do.
11	J. H. Van Ness	Irvington road, Irvington	13			do	Do.
11	H. Hedden	Irvington	4			do	Do.
11	Edward Day & Son	Irvington	65			do	
12	Mrs. Bowers	South Newark	5			do	Do.
12	A. W. Sargent	do	8			do	Do.
12	Jacob Bretz	do	4			do	Do.
12	Wm. Boshler	do	2			do	Do.
12	John Sex	do	5			do	Do.
12	John Miller	do	6			do	Do.
12	Henry Hardenfield	Irvington	19			do	Do.
16	S. Kolostonskey	Secaucus		16	Filthy	Miserable	W. B. E. Miller.
17	James Nelson	Road to Rahway, New Brunswick	36		Good	Good	Do.
18	Fredericka Aulitz	7 Pavonia avenue, Jersey City	17	5			Do.
18	Lawrence Cullen	530 West Side avenue, Jersey City	18	3			Do.
18	John Cullen	894 Montgomery street, Jersey City	11	3	Fair	Fair	Do.
19	James Martin	South Bergen avenue, Jersey City	16		Good	Good	Do.
19	Paul Stuart	do	14		do	do	Do.
19	Charles Howard	do	21		do	do	Do.
19	James Jackson	do	19		do	Fair	Do.
22	Margaret Fitzsimmons	West Side avenue, Bayonne, Jersey City	4		Fair	do	Do.
22	Wm. Mines	do	12		Good	Good	Do.
22	James Cahill	South Bergen avenue, Bayonne. Jersey City	4		do	do	Do.
22	James Lyons	Greenville avenue, Bayonne, Jersey City	22		do	do	Do.
22	Thomas Martin]	South Bergen avenue, Bayonne, Jersey City	5	1	do	do	Do.
23	F. W. Grible	Avenue C and 30th street, Bayonne, Jersey City		2			Do.
23	Sarah McIntyre	Sudick Place, Bayonne, Jersey City	4		Good	Good	Do.
23	Wm. Howard	Avenue D and Chestnut street, Bayonne, Jersey City.	3	1	do	do	Do.
23	Charles Lyons	Avenue D and Maple avenue, Bayonne, Jersey City.	23		do	do	Do.
25	Daniel Tierney	Avenue R and 2d street, Bayonne, Jersey City	14		Fair	Fair	Do.
25	Henry Baker	42d street, near Bayside avenue, Bayonne, Jersey City.	1			do	Do.
25	John Asendorf	S. Bergen avenue, Bayonne, Jersey City	1		Dirty	do	Do.
25	John Sumstein	Avenue D and, 40th street, Bayonne, Jersey City	1		Fair	do	Do.
25	Robert Grogen	George street, Bayonne, Jersey City	1		do	do	Do.
25	Jeremiah O'Harra	do	1		do	do	Do.
25	John Bunnell	do	4	1	Dirty	Very poor	Do.
25	Michael Byrne	Avenue B and 40th street, Bayonne, Jersey City	5		Good	Good	Do.
26	Henry I[illegible]ins	Chestnut avenue, Bayonne, Jersey City	15	4	Very filthy	Very poor	Do.
26	Wm. Wright	do	5				Do.
26	W. W. Waters	29th street and Avenue D, Bayonne, Jersey City	4	1	Good	Good	Do.
26	W. W. O'Neil	27th street and Avenue D, Bayonne, Jersey City	6		Fair	Fair	Do.
26	R. W. Warren	2531 South Bergen avenue, Bayonne, Jersey City	8		Good	Good	Do.
26	O. M. Taylor	2590 South Bergen avenue, Bayonne, Jersey City	5		do	do	Do.
26	H. Rider	25th street and Avenue D, Bayonne, Jersey City	1		do	do	Do.
26	Thomas Dobson	Eastside avenue, Bayonne, Jersey City	22		Very good	Very good	Do.
27	M. Stradler	47th street and Avenue C, Bayonne, Jersey City	4		Good	Good	Do.
27	D. Stegman	47th street and Bergen avenue, Bayonne, Jersey City.	2		do	do	Do.
27	V. Lauer	47th street, near Bergen ave., Bayonne, Jersey City	5		do	do	Do.
27	C. J. Dorain	47th street and Avenue C, Bayonne, Jersey City	6		do	do	Do.
27	C. Doring	46th street and Avenue D, Bayonne, Jersey City	1		do	do	Do.
27	C. M. Manoken	do	2		do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections in New Jersey—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.							
Dec. 27	L. E. Young	44th street, Bayonne, Jersey City	8		Good	Good	W. B. E. Miller.
27	James Salter	43d street, Bayonne, Jersey City	2		do	do	Do.
27	Jos. Salter	Chestnut avenue, Bayonne, Jersey City	1		do	do	Do.
27	Joseph Carroll	Pamrapo avenue, Bayonne, Jersey City	6		do	do	Do.
27	L. Van Buskirk	Chestnut avenue, Bayonne, Jersey City	8	1			Do.
27	J. C. Lewis	47th street and Avenue C, Bayonne, Jersey City	8		do	do	Do.
13	C. McMichaty	Englewood	3			do	Rowland & Hawk.
13	Capt. and Major Moore	do	5			do	Do.
18	John S. Edsal	do	10				Do.
18	J. B. Bamlien	Fairview	6			do	Do.
18	C. Hizerbottle	Englewood	14		Filthy	do	Do.
18	Saml. Degrote	Fairview	25			do	Do.
13	John Garie	do	4			do	Do.
13	Henry Davis	Englewood	9			do	Do.
13	Henry Berney	do	15			do	Do.
13	F. Freeling	Grand avenue, Englewood	10			do	Do.
13	M. A. Wood	Englewood	10			do	Do.
13	Conrad Pack	Grand avenue, Englewood	22			do	Do.
18	August Fisher	do	8			Fair	Do.
18	A. Lydecker	do	6	1		do	Do.
16	Mrs. Annie Hidie	Newark Neck	4			Good	Do.
17	Philip Manner	do	5			do	Do.
17	Joseph Fisher	do	8			do	Do.
17	Miss Essing	do	8			do	Do.
17	Joseph Smyth	do	13			do	Do.
17	Mrs. Telling	do	4			do	Do.
17	Mrs. Bowers	do	4			do	Do.
17	M. Raphael	do	3	2			Do.
17	John Aetcheck	do	3			Good	Do.
17	Patrick Doyle	do	11			do	Do.
17	C. Mason	do	8			do	Do.
17	G. Nugent	do	16			do	Do.
17	Michael Loueth	do	5			do	Do.
18	F. E. Ulitz	Marion				do	Do.
20	Morris Raphael	Newark	3	2		Fair	Do.
23	Mrs. Margarette Curley	Bloomfield (old road), Belville	4			Good	Do.
23	Mack Filey	Belville	14			do	Do.
23	Michael Roth	Grand avenue, Englewood	13			do	Do.
23	Edwin Keen	Belville	2			do	Do.

23	T. H. Decker	Bloomfield	28			do	Do.
23	Mrs. Walker	Belville	6			do	Do.
23	Martin Bergen	do	17			do	Do.
23	James Dunn	do	13			do	Do.
24	John Maxfield	Bloomfield	120		Fine	Fine	Do.
24	Fred. Sheppard	do	4			Good	Do.
24	J. W. Ostrander	East Orange	2			do	Do.
24	Mr. Mann	do	2			do	Do.
27	A. P. Brown	Milburn	43			do	Do.
27	David Carter	do	40			do	Do.
27	M. Arklerions	do	7			do	Do.
27	Jacob Eastman	Union	13			do	Do.
27	M. Johnson	do	8			do	Do.
27	James Luby	do	23			do	Do.
27	John Denley	do	4			do	Do.
30	Christian Altfelex	Guttenburg	26	10	Filthy	Filthy	Do.
30	Francis Jubbing	do	6			Good	Do.
30	Alex. McClanican	do	12			Fair	Do.
30	Peter Brown	do	7			Good	Do.

INSPECTIONS IN THE DISTRICT OF COLUMBIA.

Oct. 10	Mr. Lacey	Benning's road	36		Fair	Poor	W. H. Rose.
10	Mr. Golden	Good Hope Hill	13		Clean	Good	Do.
10	Wallace Paine	Hawley's road	26	4	Foul	Very poor	Do.
13	W. H. White	Corner Boundary and Nineteenth streets, N. W	4		Fair	Good	Do.
13	Mr. Deane	do	5		Clean	do	Do.
13	John Mahoney	do	9		Fair	Fair	Do.
13	Wm. Houtz	Near Columbia road	1	Sus.	do	do	Do.
13	Thos. Cullerton	Near Boundary road	4		Foul	Very poor	Do.
13	Mary Connor	Champagne avenue near Boundary	14			do	Do.
13	Geo. Dorr	Near Boundary, N. W	7		Clean	Fair	Do.
13	Chas. King	Columbia, N. W	8	1 rec.	Foul	do	Do.
13	Capt. C. Newbold	Sixteenth street, N. W., Mt. Pleasant	63		Fair	Poor	Do.
14	Mrs. Douohne	Twenty-fourth and Twenty-fifth streets, N. W	2		Foul	do	Do.
14	H. T. Hamilton	Twenty-fourth street bet. M and N, N. W	2		Fair	Good	Do.
14	James Newman	Hill, bet. 23d and 24th streets, N. W	3		Foul	do	Do.
14	Jas. Halman	do	1		Fair	do	Do.
14	Mrs. Flaherty	Twenty-fourth street, near M, N. W	1		do	Fair	Do.
14	A. Snubly	do	1		do	do	Do.
14	Mrs. McDonald	1920 street, N. W	3		do	do	Do.
14	Thos. Sterp	26th and M streets, N. W	2		do	do	Do.
14	John Herman	Near Rock Creek	9		Clean	Good	Do.
14	John Murphy	Twenty-first street, near Boundary	13		Fair	do	Do.
14	James Mulligan	do	12		do	Fair	Do.
14	Mrs. McKeever	1737 Twentieth street, N. W	6		Clean	Good	Do.
14	Mrs. Brown	1743 Twentieth street, N. W	2		Fair	Poor	Do.

Investigation of contagious pleuro-pneumonia—Inspections in the District of Columbia—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.							
Oct. 14	John Seibel	1916 S street, N. W	6		Clean	Good	W. H. Rose.
14	Mr. Cook	1732 Nineteenth street, N. W	5		Fair	Poor	Do.
14	Mrs. Lydon	1724 Nineteenth street, N. W	2		do	Good	Do.
14	Daniel Cady	Nineteenth street near R, N. W	2		Fair	Fair	Do.
14	Martin Noon	1712 Nineteeth street, N. W	2		Clean	Good	Do.
14	Mrs. Jas. Raferty	1917 R street, N. W	3		do	do	Do.
14	Mr. Dove	Cedar street between 17th and 18th streets, N. W	2		Foul	1 paralyzed	Do.
14	Mrs. Cresea	do	1		Fair	Good	Do.
14	Mrs. Day	1820 T street, N. W	1		do	Poor	Do.
15	Mrs. McNamee	Between Massachusetts avenue and P street, N. W	2		Foul	do	Do.
15	Mrs. O'Brien	1810 Boundary street, N. W	1	1	Clean	do	Do.
15	Mr. C. Eslin	Corner Sixteenth street and Columbia road, N. W	2		do	Fair	Do.
15	Mrs. Hamilton	Fourteenth street, N. W	20		do	do	Do.
15	Mrs. Hardon	do	14	1 rec.	Poor	Poor	Do.
15	Mrs. Blagden	Fourteenth street near Brightwood, N. W	18	2			Do.
15	Mrs. Rohr	do	2		Fair	Good	Do.
15	James White	Fourteenth street near T street, N. W	3		Clean	Fair	Do.
15	Mr. Moses	Cor. 7th and Fourteenth streets, Brightwood, N. W	2		Fair	Poor	Do.
15	Mrs. Thomas	Near 7th street, Brightwood, N. W	7		do	do	Do.
15	Mr. Carpenter	do	10		do	Good	Do.
15	Mr. F. Lay	do	7		Clean	do	Do.
15	Bennet Hatcher	do	10		Fair	Fair	Do.
15	Mr. Warner	do	3		Clean	Good	Do.
16	Mr. A. G. Mount	Corner 7th street and Soldiers' Home, N. W	28		Fair	Fair	Do.
16	"U. S."	Soldiers' Home, N. W	59		Clean	Good	Do.
16	F. Rose	Soldiers' Home road	1		Poor	Fair	Do.
16	Mr. Roberts	do	2		Fair	Good	Do.
16	John W. Brown	do	2		do	Fair	Do.
16	James H. Hollidge	Magnolia avenue	19		do	do	Do.
16	John W. Ray	do	7		do	do	Do.
16	Michael White	do	26		Foul	Poor	Do.
16	Lerb. Sterman	Magnolia avenue	5		Poor	Foul	Do.
16	Doctor White	North Seventh street	1		Clean	Good	Do.
17	Mrs. Hunter	1317 W street, N. W	1		Fair	Fair	Do.
17	Mrs. Rupert	Grant street, N. W	5		Foul	Poor	Do.
17	Mrs. B. Murphy	1945 Twelfth street, N. W	6		Clean	Good	Do.
17	Michael Keenan	2119 Eleventh street, N. W	2		Poor	Poor	Do.
17	John Driscoll	Grant street, N. W	7		do	do	Do.
17	Mrs. Newman	do	1		Foul	do	Do.

17	Mr. Lynch	do	4		do	do	Do.
17	Bridget White	Seventh street, N. W	10		do	Fair	Do.
17	Robert Barrett	Seventh street, above Boundary	9	1 rec.	do	do	Do.
17	Mrs. B. Meyers	do	7		Fair	do	Do.
17	Thos. Dunnigan	do	14		do	do	Do.
17	Mary Morrissy	do	7		Foul	Poor	Do.
17	Mrs. Alman	do	1		Clean	do	Do.
17	Dennis Murphy	Ninth street, north Boundary	11			Good	Do.
17	Pat Henry Hurley	do	14			Fair	Do.
17	Mr. McKay	do	15		Fair	Good	Do.
17	Nicholas Beck	do	4		do	do	Do.
17	Mrs. B. Keefe	do	7	1 rec.	Foul	Poor	Do.
17	Mr. Thompson	do	2		Poor	Fair	Do.
17	Mrs. Tobin	do	1		Foul	Poor	Do.
17	Leo Schmetizle	do	4		Fair	Good	Do.
17	Mr. Shugrue	do	24		Foul	Poor	Do.
17	James Owens	2116 Seventh street, Boundary	2		Clean	Good	Do.
18	Mrs. Kate McCarty	2136 Ninth street, north Boundary	2		Foul	Poor	Do.
18	Geo. Shreger	2153 Ninth street, north Boundary	1		Fair	Good	Do.
18	Mrs. Kneeland Johnson	2164 Ninth street, north Boundary	1		Foul	Poor	Do.
18	Elizabeth Federline	Seventh street, north Boundary	1		Fair	Fair	Do.
18	Mr. Bakersmith	do	1		do	do	Do.
18	Jas. McCauley	Corner Sumner avenue and Seventh street	4		Clean	Good	Do.
18	F. Westmeyer	Seventh street, opposite Schutzen Park	1		Foul	Poor	Do.
18	E. O. Scagg	do	1		Fair	Fair	Do.
18	Mrs. Connors	do	1		Foul	Poor	Do.
18	Scheutzen Park	do	2		Clean	Fair	Do.
18	Daniel Sullivan	Seventh street, north Schutzen Park	5		Foul	Poor	Do.
18	David Barry	Seventh street, near Schutzen Park	6		do	do	Do.
18	Wm. Harrison	Ninth street, north	2		do	do	Do.
18	Mrs. May	Seventh street	2		Fair	Fair	Do.
18	Mr. A. Fingle	Spring road	1		Clean	do	Do.
18	Mr. Zeabon	do	1		do	do	Do.
20	Peter Schweitzer	609 Rhode Island avenue	2		Foul	do	Do.
20	Mrs. Kate Eagan	1834 Sixth street, N. W	3		do	do	Do.
20	John Sellner	1827 Weberger street	2		do	Good	Do.
20	John Miller	Fourth street, between O and P	1		Fair	do	Do.
20	Mrs. Michael Shea	Boundary	3		Foul	Poor	Do.
20	Mrs. Kate Lucas	Pomeroy street, N. W	3		do	do	Do.
20	Martha Deane	do	1		do	do	Do.
20	Wm. Addison	do	1		do	do	Do.
20	Louis Davis	do	2		do	do	Do.
20	John Howard	do	3		Fair	Good	Do.
20	John Moore	Moore's lane	1		do	do	Do.
20	H. Daley	Fourth street, N. W	4		do	Fair	Do.
21	James Dempsey	1012 First street, N. W	7		do	do	Do.
21	Mary Bresnahan	105 L street, N. W	1		do	do	Do.
21	Mary Horan	129 L street, N. W	2		Foul	Fair	Do.
21	Mrs. C. Foley	922 First street, N. W	2		do	do	Do.
21	Mr. Drany	New Jersey avenue, N. W	1		Fair	do	Do.
21	Mr. Brooks	13 L street, N. W	3		Foul	do	Do.
21	Albert Wall	Queen Chapel road	15		do	Poor	Do.

Investigation of contagious pleuro-pneumonia—Inspections in the District of Columbia—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.							
Oct. 14	John Seibel	1916 S street, N. W	6		Clean	Good	W. H. Rose.
14	Mr. Cook	1732 Nineteenth street, N. W	5		Fair	Poor	Do.
14	Mrs. Lydon	1724 Nineteenth street, N. W	2		do	Good	Do.
14	Daniel Cady	Nineteenth street near R, N.W	2		Fair	Fair	Do.
14	Martin Noon	1712 Nineteeth street, N.W	2		Clean	Good	Do.
14	Mrs. Jas. Raferty	1917 R street, N.W	3		do	do	Do.
14	Mr. Dove	Cedar street between 17th and 18th streets, N.W	2		Foul	1 paralyzed	Do.
14	Mrs. Cresea	do	1		Fair	Good	Do.
14	Mrs. Day	1820 T street, N.W	1		do	Poor	Do.
15	Mrs. McNamee	Between Massachusetts avenue and P street, N.W	2		Foul	do	Do.
15	Mrs. O'Brien	1810 Boundary street, N.W	1	1	Clean	do	Do.
15	Mr. C. Eslin	Corner Sixteenth street and Columbia road, N.W	2		do	Fair	Do.
15	Mrs. Hamilton	Fourteenth street, N.W	20		do	do	Do.
15	Mrs. Hardon	do	14	1 rec.	Poor	Poor	Do.
15	Mrs. Blagden	Fourteenth street near Brightwood, N.W	18	2			Do.
15	Mrs. Rohr	do	2		Fair	Good	Do.
15	James White	Fourteenth street near T street, N.W	3		Clean	Fair	Do.
15	Mr. Moses	Cor. 7th and Fourteenth streets, Brightwood, N.W	2		Fair	Poor	Do.
15	Mrs. Thomas	Near 7th street, Brightwood, N.W	7		do	do	Do.
15	Mr. Carpenter	do	10		do	Good	Do.
15	Mr. F. Lay	do	7		Clean	do	Do.
15	Bennet Hatcher	do	10		Fair	Fair	Do.
15	Mr. Warner	do	3		Clean	Good	Do.
16	Mr. A. G. Mount	Corner 7th street and Soldiers' Home, N.W	28		Fair	Fair	Do.
16	"U. S."	Soldiers' Home, N.W	59		Clean	Good	Do.
16	F. Rose	Soldiers' Home road	1		Poor	Fair	Do.
16	Mr. Roberts	do	2		Fair	Good	Do.
16	John W. Brown	do	2		do	Fair	Do.
16	James H. Hollidge	Magnolia avenue	19		do	do	Do.
16	John W. Ray	do	7		do	do	Do.
16	Michael White	do	26		Foul	Poor	Do.
16	Lerb. Sterman	Magnolia avenue	5		Poor	Foul	Do.
16	Doctor White	North Seventh street	1		Clean	Good	Do.
17	Mrs. Hunter	1317 W street, N. W	1		Fair	Fair	Do.
17	Mrs. Rupert	Grant street, N. W	5		Foul	Poor	Do.
17	Mrs. B. Murphy	1945 Twelfth street, N. W	6		Clean	Good	Do.
17	Michael Keenan	2119 Eleventh street, N. W	2		Poor	Poor	Do.
17	John Driscoll	Grant street, N. W	7		do	do	Do.
17	Mrs. Newman	do	1		Foul	do	Do.

17	Mr. Lynch	do	4		do	do	Do.
17	Bridget White	Seventh street, N. W	10		do	Fair	Do.
17	Robert Barrett	Seventh street, above Boundary	9	1 rec.	do	do	Do.
17	Mrs. B. Meyers	do	7		Fair	do	Do.
17	Thos. Dunnigan	do	14		do	do	Do.
17	Mary Morrissy	do	7		Foul	Poor	Do.
17	Mrs. Alman	do	1		Clean	do	Do.
17	Dennis Murphy	Ninth street, north Boundary	11			Good	Do.
17	Pat Henry Hurley	do	14			Fair	Do.
17	Mr. McKay	do	15		Fair	Good	Do.
17	Nicholas Beck	do	4		do	do	Do.
17	Mrs. R. Keefe	do	7	1 rec.	Foul	Poor	Do.
17	Mr. Thompson	do	2		Poor	Fair	Do.
17	Mrs. Tobin	do	1		Foul	Poor	Do.
17	Leo Schmetlzle	do	4		Fair	Good	Do.
17	Mr. Shugrue	do	24		Foul	Poor	Do.
17	James Owens	2116 Seventh street, Boundary	2		Clean	Good	Do.
18	Mrs. Kate McCarty	2136 Ninth street, north Boundary	2		Foul	Poor	Do.
18	Geo. Shreger	2153 Ninth street, north Boundary	1		Fair	Good	Do.
18	Mrs. Kneeland Johnson	2164 Ninth street, north Boundary	1		Foul	Poor	Do.
18	Elizabeth Federline	Seventh street, north Boundary	1		Fair	Fair	Do.
18	Mr. Bakersmith	do	1		do	do	Do.
18	Jas. McCauley	Corner Sumner avenue and Seventh street	4		Clean	Good	Do.
18	F. Westmeyer	Seventh street, opposite Schutzen Park	1		Foul	Poor	Do.
18	E. O. Scagg	do	1		Fair	Fair	Do.
18	Mrs. Connors	do	1		Foul	Poor	Do.
18	Scheutzen Park	do	2		Clean	Fair	Do.
18	Daniel Sullivan	Seventh street, north Schutzen Park	5		Foul	Poor	Do.
18	David Barry	Seventh street, near Schutzen Park	6		do	do	Do.
18	Wm. Harrison	Ninth street, north	2		do	do	Do.
18	Mrs. May	Seventh street	2		Fair	Fair	Do.
18	Mr. A. Fingle	Spring road	1		Clean	do	Do.
18	Mr. Zeabon	do	1		do	do	Do.
20	Peter Schweitzer	609 Rhode Island avenue	2		Foul	do	Do.
20	Mrs. Kate Eagan	1834 Sixth street, N. W	3		do	do	Do.
20	John Sellner	1827 Weberger street	2		do	Good	Do.
20	John Miller	Fourth street, between O and P	1		Fair	do	Do.
20	Mrs. Michael Shea	Boundary	3		Foul	Poor	Do.
20	Mrs. Kate Lucas	Pomeroy street, N. W	3		do	do	Do.
20	Martha Deane	do	1		do	do	Do.
20	Wm. Addison	do	1		do	do	Do.
20	Louis Davis	do	2		do	do	Do.
20	John Howard	do	2		Fair	Good	Do.
20	John Moore	Moore's lane	1		do	do	Do.
20	H. Daley	Fourth street, N. W	4		do	Fair	Do.
21	James Dempsey	1012 First street, N. W	7		do	do	Do.
21	Mary Bresnahan	105 L street, N. W	1		do	do	Do.
21	Mary Horan	129 L street, N. W	2		Foul	Fair	Do.
21	Mrs. C. Foley	922 First street, N. W	2		do	do	Do.
21	Mr. Drany	New Jersey avenue, N. W	1		Fair	do	Do.
21	Mr. Brooks	13 L street, N. W	3		Foul	do	Do.
21	Albert Wall	Queen Chapel road	15		do	Poor	Do.

Investigation of contagious pleuro-pneumonia—Inspections in the District of Columbia—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
Oct. 21	John H. Harper	Bladensburg road	10		Foul	Fair	W. H. Rose.
21	Moses Stearman	do	8		Fair	do	Do.
21	Alfred Heighmuler	Old Bladensburg road	11		Poor	do	Do.
21	Mr. Moorehead	do	4		Fair	do	Do.
21	Mr. Welsh	Corner Bladensburg and Queen's Chapel roads	2		Very poor	Very poor	Do.
21	Isaac Quackenbush	Old Bladensburg road	21		Fair	Good	Do.
22	Curtis I. Gilbert	Glenwood road	12		do	do	Do.
22	Wm. Chord	do	13		do	do	Do.
22	Samuel Ceas	Old Bladensburg road	20		Foul	Poor	Do.
22	John Lacy	Glenwood road	10		Poor	do	Do.
22	Daniel Allman	do	18		Fair	Fair	Do.
22	Mrs. Emma Beale	do	7		do	do	Do.
22	Mrs. Roach	Edgewood road	19		do	do	Do.
22	Michael O'Connor	Near Glenwood road	22		do	do	Do.
22	Moses Maddy	Edgewood road	3		Poor	Poor	Do.
22	Nathan Sprague	do	12		do	do	Do.
22	Alex. McKerichar	Glenwood road	3		do	do	Do.
22	Dr. Brooks	Queenstown road	3		Fair	Good	Do.
22	Will W. Johnson	do	16		do	Fair	Do.
22	E. J. Buttington	Queen's Chapel road	8				Do.
23	Mrs. Mary Calahan	Caton's avenue, near Glenwood	1	1	Fair	Good	Do.
23	Mrs McCarthy	do	2		do	Fair	Do.
23	Moses Altman	do	4	1 rec			Do.
23	John Merritt	do	2		Fair	Fair	Do.
23	Thos. Grady	do	4		Fair	Fair	Do.
23	Pat. Cudmore	do	8		do	do	Do.
23	Mrs. Bresnahan	do	2		do	do	Do.
23	Mr. H. Hartung	Corner Prospect street, Glenwood	15		do	do	Do.
23	Mrs. Flavin	do	3	1 rec.	Poor	Poor	Do.
23	Jas. Carr	Prospect street, Glenwood	2		do	do	Do.
23	Michael Burke	do	4	1 rec.	do	do	Do.
23	John Connor	29 P street, N. W	4		do	Fair	Do.
23	Timothy Gallagher	do	2		Fair	do	Do.
23	Pat. Nelligan	Delaware avenue, corner Boundary	10		do	do	Do.
23	John Scanlon	do	2		Poor	do	Do.
23	Mrs. Johanna Alworth	Third street, between L and M	5		Fair	do	Do.
23	Cornelius Curtin	Third street, between K and L	8		do	do	Do.
23	Conrad Steurmagel	Corner Fifth and L streets	5		do	do	Do.
23	Richard Reagan	511 M street, N. E	4		do	Poor	Do.

24	Michael Bernhardt	Fifth and I streets, N. E	5		Foul	do	Do.
24	Bridget Craglan	814 Fourth street, N. E	2		Fair	Fair	Do.
24	Thos. Scanlon	1032 Fifth street, N. W	1		do	do	Do.
24	Thos. Donohue	I street, between Sixth and Seventh streets, N. E	6		do	Poor	Do.
24	F. C. Kate	806 Eighth street, N. E	1		Clean	Good	Do.
24	Joseph Palmer	1240 I street, N. E	1		do	do	Do.
24	Mrs. Burdett	1236 I street, N. E	1		Fair	do	Do.
24	Wm. Phillips	1232 I street, N. E	1		do	Fair	Do.
24	Jacob Meyers	H street, between 13th and 14th streets, N. E	1		Poor	do	Do.
24	M. Meidel	Corner Fourteenth and H streets, N. E	1	1 rec.	Foul	Very poor	Do.
24	Deaf and Dumb Asylum	Boundary, N. E	9		Fair	Fair	Do.
24	James Lewis	Bladensburg road	1		Poor	Poor	Do.
24	Mr. Spear	do	10	1 rec.	Foul	Fair	Do.
24	Little Sisters of the Poor	do	3		Clean	Fair	Do.
24	Mrs. Mulcahy	do	4		Fair	do	Do.
24	Mrs. C. McCarthy	do	5	2	Poor	Poor	Do.
24	Thos. Fenwick	Back of Mount Olivet cemetery	8		Fair	Fair	Do.
24	Theo. I. Samuels	do	5		Foul	do	Do.
24	John Boyle	Hickey's lane, near Bladensburg road	17		do	do	Do.
24	Chas. Wayhausen	do	9		do	do	Do.
25	Rudolph Frederick	Near Bladensburg road	7		do	do	Do.
25	John S. Mills	do	17		do	Poor	Do.
25	Reform School	do	13		do	do	Do.
25	Mr. Carlton	do	16	1 sick			Do.
27	James Allman	Fourteenth and C streets, S. W	11	1 sus.	Foul	Poor	Do.
27	John Hughes	924 Twenty-seventh street, S. W	3		Poor	Fair	Do.
27	J. H. Spillman	914 Twenty-sixth street, N. W	2		do		Do.
27	Wm. McCallum	Hughes alley, N. W	3		do	Poor	Do.
27	Mrs. Mary Erner	924 Twenty-fifth street, N. W	4		do	do	Do.
27	Sisters' Infant Asylum		3		Clean	Good	Do.
27	Mrs. Elizabeth Chism	731 Twenty-third street, S. W	2		Poor	Fair	Do.
27	Mr. Rhine	716 Twenty-third street, S. W	1		Foul	Poor	Do.
27	Henry Burrell	2215 D street, S. W	1		Poor	Fair	Do.
27	Mrs. Reily	Twenty-second street, S. W	2			Poor	Do.
27	John K. Pfiel	do	1		Foul	Fair	Do.
27	Mrs. Proctor	N. Y. ave., bet. Twenty-second and Twenty-third	1		do	do	Do.
27	Washington Payton	N. Y. avenue, near Twenty-first street	2		do	Poor	Do.
27	Mrs. Townby	Corner E and Twenty-first streets, S. W	1		do	do	Do.
27	Arthur Mason	427 Twentieth street, S. W	2		do	Fair	Do.
27	Mrs. Mary Fox	600 Twenty-first street, S. W	3		do	Poor	Do.
27	Mrs. M. V. Thompson	408 Nineteenth street, S. W	1		Fair	Fair	Do.
27	Miss McLaughlin	G street, between Twenty-first and Twenty-second streets, S. W.	1		Poor	Poor	Do.
27	Garrett Carter	404 Thirteen-and-a-halfstreet, S. W	1		do	do	Do.
27	Wm. Davis	425 Twelfth street, S. W	3		Foul	Fair	Do.
27	Daniel Hartney	454 Ninth street, S. W	1		Poor	Poor	Do.
27	John Whitten	Corner Four-and-a-half and P streets, S. W	3		do	Fair	Do.
27	U. S. Arsenal	Terminus Four-and-a-half, S. W	15		Clean	Good	Do.
27	Martha Paterson	413 O street, S. W	1		Fair	Fair	Do.
27	T. Long	1340 Four-and-a-half street, S. W	1		Poor	Poor	Do.
27	Thos. Connor	1216 Union street, S. W	3		do	Fair	Do.
27	Joseph McCauley	Corner Sixth and M streets, S. W	3		Fair	do	Do.

Investigation of contagious pleuro-pnuemonia—Inspections in the District of Columbia—Continued.

Date.		Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.								
Oct.	27	George Millinore	480 L street, S. W	4		Poor	Poor	W. H. Rose.
	27	Mrs. McNery	474 L street, S. W	2		do	Fair	Do.
	27	John Geyhardt	330 K street, SW	4		Foul	do	Do.
	27	Christian Betez	328 K street, S. W	1		do	Poor	Do.
	27	Gotlip Murr	1013 Third street, S. W	1		do	do	Do.
	28	Thos. Malony	485 C street, S. W	8		do	Fair	Do.
	28	Mrs. McCann	Corner D and Second streets, S. W	3		do	do	Do.
	28	Matthew Colbert	419 Second street, S. W	8		do	do	Do.
	28	John Downey	Limerick avenue, S. W	8		do	Poor	Do.
	28	Mr. Curtain	Second and D streets, S. W	1		do	do	Do.
	28	Pierre Chansky	Thirty-eighth and First streets, S. W	2		do	Fair	Do.
	28	Rachel Larrison	106 Virginia avenue, S. W	2		do	do	Do.
	28	Patrick Donohue	125 Fourth street, S. W	1		do	Poor	Do.
	28	John Sheizer	1232 Third street, S. W	2	2	Foul	Sick	Do.
	28	Mrs. Mary Maloney	125 G street, S. W	2		do	Fair	Do.
	28	Mrs. Johanna Reaghan	413 Three-and-a-half street, S. W	2		do	do	Do.
	28	Micheal Welsh	343 G street, S. W	4		Poor	do	Do.
	28	Morris Manger	704 G street, S. W	2		Foul	do	Do.
	28	Mrs. Driscoll	347 H street, S. W	3		Poor	do	Do.
	28	Michael Dunnigan	337 H street, S. W	5	1 sus.	Foul	do	Do.
	28	Mr. Curtain	612 H street, S. W	8		do	do	Do.
	28	Mr. Thos. Collins	614 H street, S. W	2		do	do	Do.
	28	Johannah Downey	621 I street, S. W	6		do	Good	Do.
	28	Mrs. Catharine Flynn	827 Sixth street, S. W	6		Fair	Fair	Do.
	28	John Flaherty	624 K street, S. W	2		Foul	do	Do.
	28	Mrs. Johannah Mahoney	628 K street, S. W	2		do	do	Do.
	28	Thos. McGrath	709 Second street, S. W	8		do	do	Do.
	28	Wm. Richards	N street near South Capitol, S. W	2	1	Fair	Poor	Do.
	28	Joseph Trapp	"T. B." road, Uniontown	20		Foul	Fair	Do.
	30	Patrick Connors	120 D street, S. E	1		do	Poor	Do.
	30	Thos. O'Brien	Eleventh and D street, S. E	1		Poor	do	Do.
	30	Susan Brown	308 Third street, S. E	1		do	Fair	Do.
	30	Mrs. James	212 D street, S. E	1		Fair	Poor	Do.
	30	Eugene Sullivan	Corner Second and E streets, S. E	2		Poor	do	Do.
	30	Providence Hospital	Corner Second and D streets, S. E	2		Clean	Good	Do.
	30	Michael Touhy	418 Third street, S. E	4		Fair	Fair	Do.
	30	Patrick Hallahan	746 Sixth street, S. E	5		Foul	do	Do.
	30	John Sullivan	311 Virginia avenue, S. E	4		do	Poor	Do.
	30	Mary Hart	816 Second street, S. E	1		Fair	Fair	Do.

	30	David Nagle	Corner Second street and Virginia avenue, S. E	2		Poor	do	Do.
	30	Abraham Haas	Corner I and First streets, S. E	1		Fair	do	Do.
	30	John Brinkley	108 M street S. E	1		do	do	Do.
	31	Francis Bailer	325 Eighth street, S. E	4		Foul	Poor	Do.
	31	Edward Bresnahan	816 C street, S. E	2		do	Fair	Do.
	31	Dennis Harrison	225 Ninth street, S. E	2		do	Poor	Do.
	31	Charles Parkes	719 Tenth street, S. E	5	1 rec.	do	do	Do.
	31	Mr. E. B. Hughes	Corner Eighth and D streets, N. E	1		Fair	Fair	Do.
	31	Mrs. Mary Obenstein	Corner Eleventh and D streets, N. E	4	1 rec.	Foul	Poor	Do.
	31	Bridget Reily	Massachusetts avenue and B streets, N. E	2		Fair	Fair	Do.
	31	Wm. George	619 B street, N. E	2		do	do	Do.
	31	G. W. Williamson	609 B street, N. E	2		do	Good	Do.
	31	Thos. Healy	617 A street, N. E	2		Poor	Fair	Do.
	31	Thos. Noone	22 Fifth street, N. E	2		Fair	do	Do.
	31	Mrs. Clancy	18 Fifth street, N. E	2		do	do	Do.
	31	Mrs. Catharine Lynch	11 Seventh street, N. E	3		Foul	do	Do.
	31	Mrs. Ellen Flynn	105 Eleventh street, N. E	4		do	do	Do.
	31	Wm. H. Tufts	1003 North Carolina avenue, N. E	8		Clean	Good	Do.
	31	U. S. Navy Yard	Foot Eighth street, N. E	8		do	Fair	Do.
	31	Wm. Vanderslie	1235 Tenth street, N. E	2		Fair	do	Do.
	31	Samuel Langley	1206 Eighth street, N. E	1		Poor	do	Do.
	31	Mrs. Mary Hogan	1216 I street, N. E	4		do	do	Do.
	31	Allen & Webster	C street between Eleventh and Twelfth streets, N. E	4		Foul	Poor	Do.
	31	Mrs. Cunningham	C street between Tenth and Eleventh streets, N. E	1		Poor	Fair	Do.
	31	James Kalicher	810 D street, N. E	8		Fair	do	Do.
Nov.	1	Mrs. Heiss	G street corner Thirteenth street, N. E	1		do	do	Do.
	1	Frank Godfrey	Seventh street, N. E	8		Poor	do	Do.
	1	Thos. Ready	Corner Seventh and H streets, N. E	4		do	do	Do.
	1	J. H. Buscher	Corner Twelfth and E streets, N. E	1		Fair	do	Do.
	1	Peter Miller	D st. between Thirteenth and Fourteenth sts., N. E	8		do	do	Do.
	1	John Guethler	do	1		Poor	do	Do.
	1	Albert Dowdy	Near Fifteenth street and Pennsylvania avenue, N. E	16				Do.
	1	Mrs. Elizabeth Flynn	A st. between Fourteenth and Fifteenth sts., N. E	4	1	Foul	Poor	Do.
	1	John Crawford	Seventeenth street near East Capitol street, N. E	9	1 rec.			Do.
	1	Patrick Glancy	E st. between Sixteenth and Seventeenth sts., N. E	4	Sus.	Poor	Fair	Do.
	1	Mr. Bartlett	E street, bet. Thirteenth and Fourteenth, S. E	1		Poor	Fair	Do.
	1	Mrs. Herrill	Cor. Thirteenth street and Pennsylvania avenue, S. E	1			do	Do.
	1	Mrs. Rosenberg	Corner Tenth and C streets, S. E	1			do	Do.
	1	Mr. Dodge	Near Tenth and C streets, S. E	1			do	Do.
	1	George Clarke	Cor. Fourteenth street and Pennsylvania avenue, S. E	8		Fair	do	Do.
	1	Bernhardt Greenfeldt	Corner Thirteenth and L streets, S. E	3		Poor	Poor	Do.
	1	Mrs. Knalley	Corner Twelfth street and Georgia avenue, S. E	2		do	Fair	Do.
	1	L. M. Hummer	1207 K street, S. E	2		do	do	Do.
	1	R. J. Prather	1112 K street, S. E	1		Fair	do	Do.
	1	Richard Horn	A street, S. E., near jail	7		Foul	do	Do.
	1	Mrs. Johanna Bensinger	A street, bet. Sixteenth and Seventeenth, S. E	8		do	do	Do.
	1	Richard Walker	Seventeenth street, near East Capitol street, S. E	3		do	Poor	Do.
	1	Thos. McEntyre	1320 B street, N. E	2		do	Fair	Do.
	1	John Schwarzen	1304 D street, S. E	3	1 Sus.	do	Poor	Do.
	3	Mrs. F. Killigan	717 Third street, N. W	1		Fair	Fair	Do.
	3	Mrs. J. Magnahan	615 Second street, N. W	2		Poor	do	Do.
	3	Mrs. Kate Foley	29 F street, N. W	2		do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections in the District of Columbia—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.							
Nov. 3	Mrs. J. Driscoll	31 F street, N. W	2		Poor	Fair	W. H. Rose.
3	Mrs. P. Collins	28 Massachusetts avenue, N. W	2		Foul	do	Do.
3	John Burns	25 Massachusetts avenue, N. W	4		Poor	do	Do.
3	Denis Fitzgerald	14 G street, N. W	2		do	do	Do.
3	Michael O'Brien	30 G street, N. W	2		do	do	Do.
3	Morris Collins	39 F street, N. W	1		do	do	Do.
3	Matthew Sexton	Corner Jackson alley and North Capitol street, N. W.	5		do	do	Do.
3	Mrs. W. Caton	Jackson alley, N. W	2		Foul	do	Do.
3	Mr. L. Cullinane	do	3		do	Poor	Do.
3	Mrs. Mary Powell	do	2		do	do	Do.
3	Mrs. A. Sherdy	do	8		Poor	Fair	Do.
3	Henry Ready	32 G street, N. E	8		Poor	Poor	Do.
3	John Connell	Jackson alley, N. E	7		Fair	Good	Do.
3	James Lyons	First street, near H street, N. E	5		Poor	Fair	Do.
3	Mrs. H. Mahoney	118 G street, N. E	2		Fair	do	Do.
3	Mrs. Mary Farrell	200 G street, N. E	8		do	co	Do.
5	Mary O'Brien	403 Second street, N. E	3		Foul	do	Do.
5	Thomas Welsh	405 Second street, N. E	1		do	do	Do.
5	Mary Green	216 E street, N. E	1		Poor	Poor	Do.
5	Mrs. Sweeney	702 Second street, N. E	1		do	Fair	Do.
5	W. Brahler	Corner Fourth and H streets, N. E	2		Fair	do	Do.
5	Patrick Connell	715 Fourth street, N. E	2		Foul	Poor	Do.
5	Catharine Canty	519 H street, N. E	2		Fair	Fair	Do.
5	John Leppard	G street, between Sixth and Seventh streets, N. E	1		do	do	Do.
5	F. A. Godfrey	724 Seventh street, N. E	2		do	Poor	Do.
5	Ferdinand Bogia	621 Tenth street, N. E	8		Poor	Good	Do.
5	Mr. Schwartz	Corner Tenth and H streets, N. E	1		Fair	Fair	Do.
5	Mrs. Wilson	1237 F street, N. E	5		Poor	Poor	Do.
5	Thos. Hannahan	Benning's road, N. E	8	1 rec.	Fair	do	Do.
6	Mrs. Flannagan	Near Benning's road, N. E	11	1 rec.	Poor	do	Do.
6	Wm. Bradley	do	9		Fair	do	Do.
6	Patrick Clancy	do	3		Poor	do	Do.
6	James Flaherty	Fourteenth street, near H street, N. E	6	2	do	do	Do.
6	Patrick Connors	Fifteenth and D streets, N. E	21		Clean	Fair	Do.
6	District jail	N. E	1		do	Good	Do.
6	Alms-house	Near Eastern Branch, N. E	13		do	do	Do.
6	J. Frank Jones	Nineteenth and C streets, N. E	4		Poor	Fair	Do.
17	James Foley	Near Benning's road, N. E	9		do	Poor	Do.

17	Mr. Brown	Near Veterinary station, N. E	8	1 rec.			Do.
7	Mrs. Bennett	Benning's road, N. E	2		do	Fair	Do.
7	John Mason	do	12		do	Poor	Do.
7	Mrs. B. Downey	Jackson street, Uniontown	3		do	Fair	Do.
7	Marden King	Harrison street, Uniontown					Do.
7	Wm. King	Sumter street, Uniontown	2		Fair	Fair	Do.
7	Allen Anderson	Harrison street, Uniontown	2		Poor	do	Do.
7	Mr. Haines	do	1		Fair	do	Do.
7	Peter Guigon	Fillmore street, Uniontown	4		do	Poor	Do.
7	F. Allen	do	1		do	Fair	Do.
7	Geo. F. Pyles	Harrison street, Uniontown	3		do	do	Do.
7	Mrs Joseph Kain	Monroe street, Uniontown	1		do	do	Do.
7	Mrs. Perkins	do	1		do	do	Do.
7	John C. Shea	Nichols avenue	8		Clean	do	Do.
7	Mr. Sherwood	do	2		do	do	Do.
7	John Liverpool	do	2		Poor	do	Do.
7	Gill Tolliver	do	1		do	do	Do.
7	Saint Elizabeth I. A	do	195		Very clean	Good	Do.
7	Samual A. Smith	Stantontown hard road	7		Poor	Fair	Do.
7	Frank Waller	Hawley's road	37		do	do	Do.
7	Henry Hawley	Nichols avenue	18		New	Very poor	Do.
7	Wallace Pain	Hawley's road	1		Fair	Fair	Do.
8	Mr. H. Robey	Harrison street, Uniontown	1		do	do	Do.
8	T. J. Anderson	do	1		do	do	Do.
8	J. H. Anderson	do	1		do	do	Do.
8	Mr. Smith	do	1		do	do	Do.
8	Mr. Thomas	River road	2		do	do	Do.
8	Mr. Grimes	do	2		Clean	do	Do.
8	German Orphan Asylum	Good Hope Hill, S. E	4		Fair	Poor	Do.
8	Phil. Simpson	"T. B." road, near Hamilton road	14		do	Fair	Do.
8	Jeremiah Dorsey	Hamilton road	5		Foul	Poor	Do.
8	John R. Catlen	Wheeler road	7		Fair	Fair	Do.
8	Joseph Waller	do	24		do	do	Do.
8	Mrs. Gozenbach	Benning's road	2		do	do	Do.
8	Mr. Rupert	do	1		do	do	Do.
8	Mrs. John Douglass	do	2		Clean	do	Do.
8	Mr. Dorr	Near Veterinary station	1		Fair	do	Do.
10	Jacob Kinally	Tenallytown road	1		do	do	Do.
10	Nicholas Connelly	do	8		Poor	do	Do.
10	Thos. Feeny	do	6		do	do	Do.
10	Wm. McLean	do	2		Fair	Good	Do.
10	Geo. Kenneally	do	2		do	Fair	Do.
10	College Villa	do	9		Poor	do	Do.
10	Louis Means	Lufborough road	4		Fair	do	Do.
10	John Donohue	do	7		Poor	do	Do.
10	Capt. Wm. Thompson	do	10		Fair	Good	Do.
10	Mr. E. Andrews	Mill road, Tenallytown	21		Poor	Poor	Do.
10	L. P. Hazel	Lufborough road, Tenallytown	4		Fair	Fair	Do.
10	Godfrey Goebel	do	2		do	do	Do.
10	Joseph Davis	do	29		Poor	do	Do.
10	Mr. Rosenbush	do	4		Fair	do	Do.
10	J. Harrington	do	31		do	do	Do.

Investigation of contagious pleuro-pneumonia—Inspections in the District of Columbia—Continued.

Date.	Name of owner or person in charge.	Location of stables and herds.	Number of cattle.	Number with lung plague.	Condition of stable.	Condition of animals.	Name of inspector.
1884.							
Nov. 10	Samnel Burrows	River road, Tenallytown	20		Poor	Good	W. H. Rose.
10	Fred. Bengerter	Fort Reno, Tenallytown	21		do	Poor	Do.
10	Mrs. Nurse	Tenallytown	6		Clean	Fair	Do.
13	John Harrigan	1320 Thirty-sixth street, Georgetown	5		Poor	do	Do.
13	Mrs. Catharine Hurley	3619 O street, Georgetown	2		Fair	do	Do.
13	Patrick Connors	3622 O street, Georgetown	1		Fair	Fair	Do.
13	Mr. Lynch	3621 O street, Georgetown	1		do	do	Do.
13	Georgetown College	Georgetown	17		do	do	Do.
13	Mrs. Maher	3519 O street, Georgetown	2		do	do	Do.
13	Daniel O'Connor	Georgetown	3		do	do	Do.
14	Louis Chisley	2543 O street, Georgetown	3		Poor	Poor	Do.
14	John Brislahan	1673 High street, Georgetown	3		do	do	Do.
14	Andrew Thurer	1667 High street, Georgetown	2		Fair	Fair	Do.
14	John Collahan	1820 Market street, Georgetown	7		Poor	do	Do.
14	Peter Moreland	3327 Fifth street, Georgetown	2		do	do	Do.
14	Academy of Visitation	Fayette street, Georgetown	14	1			Do.
14	John Callahan	48 Sixth street, Georgetown	6		Poor	Fair	Do.
14	Mrs Annie Scior	3146 Dumbarton avenue, Georgetown	2		do	Poor	Do.
14	John May	3136 Dumbarton avenue, Georgetown	3		do	do	Do.
15	John Rick	Canal road, Georgetown	15		do	do	Do.
15	John Brown	Canal and Conduit roads, Georgetown	1		do	Fair	Do.
15	Patrick Finley	Conduit road, Georgetown	1		Fair	do	Do.
15	Thos. Sullivan	do	13		Poor	Poor	Do.
15	Timothy O'Neil	do	2		do	Fair	Do.
15	Mr. S. Swindells	do	1		do	Poor	Do.
15	Henry Smith	do	1		Fair	Fair	Do.
15	Thos. Ready	do	20		do	Good	Do.
15	Mr. V. Wifbong	do	4		do	Fair	Do.
15	Wm. H. Allder	do	19		do	do	Do.
15	James Shugrue	do	13		Poor	Poor	Do.
15	Charles McCarty	do	13		do	do	Do.
15	Mrs. John Weaver	do	10		Fair	Fair	Do.
15	Robert Weaver	do	19		do	do	Do.
15	Patrick Henry Hurley	Corner Fourteenth and H streets	14		do	do	Do.

RECAPITULATION.

Location.	Number of stables and herds examined.	Number of cattle examined.	Number of animals affected with contagious pleuro-pneumonia.	Number of herds infected.
New York City	1,466	3,031	53	20
Long Island, N. Y	1,367	9,770	327	119
Staten Island, N. Y	455	3,875	13	12
New Jersey	442	5,719	131	72
District of Columbia	423	2,496	31	26
REINSPECTIONS.				
Brooklyn	60	868	100	
New Jersey	4	89	11	
Total	4,217	25,798	666	249

INSPECTION OF SLAUGHTER-HOUSES AND OFFAL-DOCK.

In addition to the above, visitations were made to the slaughter-houses and offal-dock of New York, with the following results:

Number of visits to slaughter-houses and offal-dock	333
Number of animals and carcasses inspected	722
Number affected with chronic and acute pleuro-pneumonia	68

Forty visits were also made to the Jersey City abattoir, which resulted in the finding of thirteen carcasses diseased with contagious pleuro-pneumonia.

EXPERIMENTS ON THE CONTAGIOUSNESS OF PLEURO-PNEUMONIA IN THE UNITED STATES.

During the year 1883, 15 head of cattle, consisting of calves, steers, and cows, which had been purchased in the vicinity of Washington, were exposed from time to time to sick cows taken from the dairies of the District of Columbia which presented the symptoms of contagious pleuro-pneumonia. With the exception of one heifer, none of these showed any symptoms of the disease, although they were kept under observation for more than twice the period of incubation of this plague. The heifer referred to contracted a severe cough with slight symptoms of lung affection, but there was no positive evidence of pleuro-pneumonia.

It is a well-known fact that the inoculation into the cellular tissue of the exudation liquid of contagious pleuro-pneumonia produces an intense inflammation of a progressive character which generally terminates fatally; while the same liquid from ordinary inflammations of the lungs when similarly inoculated produces no noticeable results. We have here, therefore, another means of obtaining evidence as to the nature of the lung disease of cattle which is found in this country. Accordingly, on September 22, 1883, three animals were inoculated by hypodermic injection into the cellular tissue of the neck of one drachm of exudation liquid from a cow which had just died in the acute stage of pleuro-pneumonia. The post-mortem examination of this cow demonstrated that the lungs were adherent to the ribs and diaphragm, that the cavity of the thorax contained a large quantity of clear, yellowish effusion, and that the lung tissue was extensively hepatized, red and marbled in appearance, and the interlobular connective tissue distended with exudation liquid.

A small swelling formed at the point of inoculation with each of these animals, which gradually increased in size and sensitiveness, extending downwards and backwards between the fore legs and beneath the sternum.

October 10, one of them, a two-year-old steer, was found dead in his stall. An enormous swelling occupied the region of the neck, sternum, and even extended along the sides and behind the shoulders. On cutting across this swelling it was found to be formed by the exudation of straw-colored serum into the spaces of the cellular tissue. These spaces were greatly distended, the serum flowed from them in a considerable quantity when a section was made, and the tissue was thickened and fibrous.

October 19 a second one of the inoculated animals was suffering so severely that it was slaughtered. This steer had a very large swelling in front of the shoulder and extending under the sternum. The skin had become gangrenous and sloughed from this over a space of about 4 inches square, and from the exposed surface there was an abundant discharge of pus. The swelling consisted, as in the animal which had previously died, of fibrous connective tissue, the spaces of which were distended with a clear, almost colorless exudation liquid. There were a few collapsed lobules in the lungs, and an ounce, more or less, of straw-colored serum in the pleural cavity.

The third animal inoculated had a considerable swelling at the point where the virus was introduced, but was much less severely affected than the two just referred to, and, finally, entirely recovered.

The very severe effects produced by this inoculation indicated very strongly, therefore, that the cow from which the serum was obtained was affected with contagious pleuro-pneumonia and not with an ordinary sporadic inflammation of the lungs and pleuræ.

The appearance of the lungs of the sick animals found affected with pleuro-pneumonia in this country, and the results of these inoculation experiments, taken with the history of the disease in various stables, left little reason to doubt that the pleuro-pneumonia of the United States was identical with the contagious pleuro-pneumonia or lung plague of Europe. The animals which had been exposed by cohabitation with sick cows had shown a surprising resistance to the contagion, and a much greater degree of insusceptibility than we had any reason to expect. It now became a question if these experimental animals had not been exposed to the disease and recovered from a mild attack before we obtained them. At all events, it is known to be a fact that the animals of any district in which a contagious disease has existed for a long time become much more insusceptible to that disease than are the animals of localities which have never been infected. With this fact in mind, four cows were purchased in Charlestown, W. Va., and taken to the experimental station on March 19, 1884.

The four cows from West Virginia were placed in a stable that had been previously occupied by cows affected with lung disease, and from May 1 to May 5 they were exposed to a cow from a Washington dealer which was affected with a very severe attack of inflammation of the lungs and pleuræ.

May 23, cow No. 1 had a severe cough with a harsh respiratory murmur over the right lung and a temperature of 105⅕°. May 26 there was loss of appetite, diminished milk secretion, a more distressing cough, and the temperature was still elevated. These symptoms continued with diarrhea and rapid loss of flesh until June 5, when she was destroyed in the last stages of the disease. The breathing at this time was labored and painful; there was dullness on percussion over the right side of the thorax and over the superior portion of the left side, and a loss of respiratory murmur over the same areas.

On *post-mortem* examination, the anterior lobe of the right lung was found normal, but the posterior two-thirds was hepatized, marbled, and the connective tissue bands widely distended with lymph. The pleura was greatly thickened and solidly attached to the ribs and diaphragm. The lesions in the lung were of different stages of development; some of the lobules presented a dark red infarction, others light red hepatization, while still others had not yet passed through the period of active congestion. The left lung contained in its posterior third a hepatized portion about six inches in diameter, which on section showed the same marbled appearance, and the interlobular connective tissue was distended with exudation as in the other lung. On this side adhesions had not yet formed.

Cow No. 2 had a slight cough June 1, with total loss of appetite and suspension of milk secretion. There was dullness over the left lung and a temperature of 105°. June 10, these symptoms were complicated with diarrhea and great prostration. June 12, the animal commenced eating soft food, and there was slight improvement in general appearance. June 17, she aborted and had inversion of the uterus, but under proper treatment this accident was recovered from, and she continued to gain strength until August 4, when she was slaughtered. The cough and dullness over the lung as well as an abnormally high temperature continued to the time of slaughter.

At the autopsy, the left lung was found solidly attached by false membranes to the ribs and diaphragm. The posterior part of this lung contained a large encysted mass of hepatized lung tissue, about four by six inches in size. The surrounding parts, embracing about two-thirds of the lung, had undergone fibrous degeneration, and were no longer capable of inflation. The right lung was not affected.

Cow No. 3, on May 25, had a severe cough and dullness over a part of the left lung, with increased respiratory murmur. There was no increase in temperature, and the appetite continued good. When slaughtered, August 4, there were no symptoms of disease beyond the cough, dullness on percussion, and modified respiratory sound.

The autopsy revealed the fact that the right lung was attached to the ribs and diaphragm by pleural adhesions, and the left lung was also attached over a surface three or four inches square. A large tumor was found in the thorax, which had evidently resulted from inflammation of a lymphatic gland, and closely resembled liver tissue in appearance. There was no indication of hepatization or other changes in the lung tissue.

Cow No. 4, on June 9, had a temperature of 103½°. By June 14 there was loss of appetite, suspension of milk secretion, and a temperature of 104°, with dullness over the upper part of the right lung. She gave birth to a live calf June 16, at full term, and from this time continued to improve, though the cough and lung lesion were not noticeably affected.

When slaughtered August 4 the right lung was found adherent to the ribs in its upper posterior portion corresponding to the area of dullness. On section of the lung at this point a small encysted mass of hepatized lung tissue about two inches across was found. This was the extent of the lesions in the lungs. There was a small quantity of pleural effusion on the right side, and also a considerable amount of liquid in the pericardium.

It is evident from the above statement of the symptoms and post-mortem appearances that not one of these cows escaped infection. The disease to which they were exposed was consequently contagious pleuro-pneumonia. It is to be remarked that in three of the four cases the disease was so mild that it would not have proved fatal, and that in one case there was no hepatization of the lung tissue, the lesions being limited to the pleura. Such cases have been frequently recorded in Europe, and I have met with a number of them in this country, but there has been a disposition among American veterinarians to consider hepatization and distension of the interlobular connective tissue as the essential characters of contagious pleuro-pneumonia. It is plain, however, that if all cases where these characters are wanting are pronounced to be of sporadic origin some cases of genuine lung plague would be considered as a harmless form of disease, and the proper measures for its control would consequently be neglected.

EXPERIMENTS ON BARREN ISLAND, NEW YORK.

To test the contagiousness of this disease as it exists in Brooklyn, N. Y., an experiment on a much larger scale was begun in September, 1884, at a new stable erected for the purpose on Barren Island. An attempt was first made to locate this stable on the main land of Long Island, in Brooklyn or its vicinity; but such objections were raised to this that Barren Island was selected because of its isolation. The few cattle that were kept on the island for the use of the residents had never been affected with a disease of this nature, and an ample plot of ground was obtained at a sufficient distance from the rendering works to make it impossible that disease could be contracted in any way from that establishment. Every precaution was observed to make this experiment positive and trustworthy, and I believe the results can be accepted with implicit confidence. I myself witnessed the *post-mortem* examination of three of the experimental animals, which were affected in both lungs and presented the characteristic lesions which are accepted the world over as peculiar to the contagious pleuro-pneumonia of Europe.

The results of this experiment are very convincing, 22 out of 31 animals, or 71 per cent. of those exposed, contracting the disease between September 20 and January 3, or within about three and one-half months.

The following report of Dr. McLean, who was in charge of this stable, gives a more detailed account of the experiment:

BROOKLYN, N. Y., *January* 5, 1885.

SIR: I herewith beg to report the results of the experiments, as conducted on Barren Island, under your instructions, to test the contagious or non-contagious nature of pleuro-pneumonia as met with in cattle in this country, and to decide as to its identity with the zymotic contagious pleuro-pneumonia met with in Europe, many stock-owners and a few professional men in this country being skeptical upon these points.

Your selection of Barren Island was one perfectly adapted for this purpose from its isolated position, and the fact that the fifteen native cattle thereon were never known to be affected with any contagious disease. New stables were erected to accommodate twenty-five cows and thirteen calves, and on the 14th September, 1884, eighteen cows and thirteen calves were obtained direct from Canada, a country in which contagious pleuro-pneumonia has not yet appeared, and, after a careful and satisfactory examination as to their healthy condition, were placed in the stable, into which were introduced five cows selected in the city of Brooklyn as being affected with contagious pleuro-pneumonia, with the results as exhibited in the annexed tabular statement, showing that on the 3d January, 1885, when the last were slaughtered, fourteen out of the eighteen had contracted contagious pleuro-pneumonia, and on *post-mortem* examination exhibited the specific lesions of that disease, and that in the instance of the four cows, numbers 18, 2, 15, and 5, slaughtered in the presence of yourself, Dr. Miller, and General Vernon, upon the 29th October, 1884, number 18 had, after a cohabitation of only seventeen days with the diseased animals, exhibited a temperature of 105° F., and on the twenty-first day manifested on physical examination the characteristic signs of contagious pleuro-pneumonia, and that number 15 showed the same symptoms respectively on the twentieth and twenty-seventh days.

The pathological pulmonary lesions exhibited on the *post-mortem* examination of these four were those of acute contagious pleuro-pneumonia, showing the pathognomonic marbled appearance peculiar to that disease, and, as far as pathology goes, were typical of the others which were slaughtered at a similar stage.

Among thirteen calves introduced on the 14th September, 1884, the first case developed itself on the 12th November; the next on 25th November, followed by another on the 26th November, and eventually out of the entire number up to the date of slaughter, the 2d instant, but five remained healthy.

Thus, out of a total number of thirty-one healthy animals, twenty-two contracted contagious pleuro-pneumonia between the dates of 20th September, 1884, when the first diseased animals were introduced, and the 3d January, 1885, when all remaining alive were slaughtered, establishing beyond a doubt its contagious character and its identity with the disease which has carried such havoc among the herds of Europe, and with which a long experience in Scotland has rendered me perfectly familiar.

I may say that the animals were liberally fed upon hay, bran, and oil meal, and very comfortably housed.

Respectfully, your obedient servant,

L. McLEAN,
M. R. C. V. S.

Dr. D. E. Salmon,
Chief of Bureau of Animal Industry.

No.	History.	Date of entry.	Temperature when admitted.	First abnormal temperature.	Date of first abnormal temperature.	Lung lesions detected.	Killed.	Remarks.
			° *Fahr.*	° *Fahr.*				
I.	Canadian cow, 5 years old, 1 month calved.	Sept. 14, 1884....	101	103	Nov. 14, 1884....	Nov. 22, 1884....	Jan. 2, 1885......	Chronic lesions; one-third of left lung involved.
II.	Canadian cow, 4 years old, 6 weeks calved.	Sept. 14, 1884....	101	102⅕	Oct. 21, 1884.....	Oct. 29, 1884.....	Oct. 29, 1884.....	Lungs commencing to show primary hepatization.
III.	Brooklyn cow, affected with pleuro-pneumonia.	Sept. 29, 1884....	106	106	Sept. 29, 1884....	Sept. 29, 1884....	Oct. 1, 1884......	Both lungs acutely affected, one weighing 30 pounds; large quantity of effusion.
IV.	Canadian cow, 8 years old, 2 months calved.	Sept. 14, 1884	101⅕	105½	Nov. 2, 1884	Nov. 14, 1884	Nov. 19, 1884	Lung lesions specific; one lung weighed 38 pounds; exhibited to a committee of veterinarians at Fifth Avenue Hotel.
V.	Canadian cow, 4 years old, 1 month calved.	Sept. 14, 1884....	102	103⅕	Oct. 12, 1884.....	Oct. 18, 1884.....	Oct. 29, 1884.....	Both lungs involved, showing hepatized lesions of contagious pleuro-pneumonia.
VI.	Canadian cow, 9 years old, 2 months calved.	Sept. 14, 1884....	102	104½	Dec. 7, 1884	Dec. 9, 1884	Jan. 3, 1885	Lungs showed broken-down encysted mass.
VII.	Canadian cow, 8 years old, 1 month calved.	Sept. 14, 1884 ...	101⅕	103½	Nov. 14, 1884	Nov. 18, 1884....	Jan. 2, 1885......	Limited but specific lesions of contagious pleuro-pneumonia.
VIII.	Brooklyn cow, affected with contagious pleuro-pneumonia.	Sept. 20, 1884....	103	103	Sept. 20, 1884....	Sept. 20, 1884....	Jan. 2, 1885......	Specific encysted mass of left lung of contagious pleuro-pneumonia, weighing 6 pounds.
IX.	Canadian cow, 8 years old...	Sept. 14, 1884....	101⅗	104	Oct. 23, 1884.....	On post-mortem.	Jan. 2, 1885......	Characteristic encysted portion, size of an apple, found anterior lobe of left lung.
X.	Canadian cow, 4 years old, 6 weeks calved.	Sept. 14, 1884....	102	None	None	None	Jan. 2, 1885......	Lungs normal.
XI.	Canadian cow, 6 years old, 2 months calved.	Sept. 14, 1884 ...	102	105	Oct. 28, 1884.....	None	Jan. 2, 1885......	Do.
XII.	Brooklyn cow, affected with contagious pleuro-pneumonia.	Sept. 20, 1884....	102	102	Sept. 20, 1884....	Sept. 20, 1884....	Jan. 2, 1885......	Chronic lung lesions when admitted.
XIII.	Canadian cow, 5 years old, 2 months calved.	Sept. 14, 1884 ..	102	103	Oct. 21, 1884.....	None	Jan. 2, 1885......	Lungs normal.
XIV.	Canadian cow, 5 years old, 1 month calved.	Sept. 14, 1884....	101⅗	104	Nov. 16, 1884....	Nov. 21, 1884	Jan. 2, 1885......	Almost entire right lung involved; contagious pleuro-pneumonia.
XV.	Canadian cow, 6 years old, 3 weeks calved.	Sept. 14, 1884....	102	104	Oct. 9, 1884	Oct. 15, 1884.....	Oct. 29, 1884.....	Both lungs characteristically affected with contagious pleuro-pneumonia and extensive pleuritic complications.
XVI.	......do	Sept. 14, 1884....	101⅗	104	Nov. 16, 1884	Nov. 19, 1884	Jan. 3, 1885......	One lung showed extensive chronic lesions of contagious pleuro-pneumonia.

XVII.	Brooklyn cow, affected with contagious pleuro-pneumonia.	Sept. 26, 1884	105⅕	105⅕	Sept. 26, 1884	Sept. 26, 1884	Oct. 5, 1884*	
XVIII.	Canadian cow, 9 years old, 2 weeks calved.	Sept. 14, 1884	102	105	Oct. 6, 1884	Oct. 10, 1884	Oct. 29, 1884	Lungs exhibited specific lesions of contagious pleuro-pneumonia.
XIX.	Canadian cow, 6 years old, 3 weeks calved.	Sept. 14, 1884	101	103	Nov. 10, 1884	Nov. 19, 1884	Jan. 3, 1885	Lungs showed limited but characteristic lesions of contagious pleuro-pneumonia.
XX.	Canadian cow, 8 years old, 6 months calved.	Sept. 14, 1884	101⅗	104	Oct. 28, 1884	Nov. 1, 1884	Nov. 4, 1884	Extensive lesions of contagious pleuro-pneumonia; both lungs weighed 32 pounds.
XXI.	Canadian cow, 4 years old, 6 months calved.	Sept. 14, 1884	101⅕	105	Nov. 10, 1884	Nov. 14, 1884	Dec. 3, 1884*	Right lung completely involved; contagious pleuro-pneumonia.
XXII.	Canadian cow, 5 years old, 6 months calved.	Sept. 14, 1884	101⅕	None	None	None	Jan. 2, 1885	Lungs normal.
XXIII.	Brooklyn cow, affected with contagious pleuro-pneumonia.	Oct. 25, 1884	105	105	Oct. 25, 1884	Oct. 25, 1884	Dec. 2, 1884*	

*Died.

EXPERIMENTS WITH WEST VIRGINIA CATTLE.

At the time the test was in progress at Barren Island another experiment was going on at the station in Washington. Six cows were brought from West Virginia on August 18 and placed in a stable where sick cows had previously been kept. They remained in this stable without being exposed to any sick cows until November 1, and showed no symptoms of disease with the exception of a slight cough, from which they entirely recovered.

Sick cows, presenting the symptoms of pleuro-pneumonia, from three different stables in the vicinity were introduced among these healthy cows on November 1, November 8, and November 13. On December 11 No. 1 commenced to cough; on December 14 her temperature was 103⅕°; on December 15 there was dullness over the left lung; December 21 there was loss of appetite, emaciation, diarrhea, and a temperature of 104⅗°. This animal died January 5, 1885. The autopsy revealed adhesions between the left lung and ribs, effusion of liquid into the pleural cavity. The pericardium was thickened and had become hard and fibrous. The lung tissue itself was not greatly changed, and there was no appearance of hepatization.

Cow No. 2 had a temperature of 103⅖° December 14. Two days later she had the characteristic cough of pleuro-pneumonia with dullness over the left lung. December 20 her temperature registered 105°, and the milk secretion was suspended. December 24 there was diarrhea and depression which ended in death January 9. On autopsy the left lung was found to weigh 33¾ pounds; two-thirds of the organ was completely hepatized; there was plain marbled appearance and all stages of disease from simple congestion to gray hepatization. The pleura was greatly thickened and adherent to ribs and there was about 1½ gallons of liquid effusion in the pleural cavity.

Cow No. 3 commenced coughing December 17; December 19 her temperature was 105⅖°, and the following day there was dullness over the right lung. December 22 loss of appetite, and milk secretion suspended. December 25 there is slight improvement in condition, breathing is easier; there is some appetite, and temperature has dropped to 103°. January 7 there is no further change; the milk secretion has not returned, and the dullness over the right lung remains.

Cow No. 4 began coughing December 23, and on December 25 there was a temperature of 104⅖°, and dullness on percussion over the left lung. There has been no loss of appetite, and the milk secretion continues though diminished in quantity. At the latest examination, January 7, 1885, the dullness on percussion remains, the cough continues, the respirations are hurried, and the temperature is still 105°.

Cows Nos. 5 and 6 cough continually, their appetite remains good, and their temperature has been normal. The milk secretion of No. 5 is diminished.

The result of all these experiments may be tabulated as follows:

Where experiments were made.	Number exposed.	Number certainly affected.	Number probably affected.
First experiment at Washington	15	....	1
Second experiment at Washington	4	4	....
Third experiment at Washington	6	4	2
Experiment at Barren Island	31	22	....
Total	56	30	3

That is, of the whole number exposed, 53.5 per cent. certainly contracted pleuro-pneumonia, and an additional 5 per cent. had symptoms indicating a mild attack of the same disease. If, however, we leave out of consideration the first lot of animals, which were plainly insusceptible to this disease, we find that 30 animals out of 41 contracted it by exposure, a proportion of 73 per cent. This proportion is much greater than is usually seen with this disease in Europe, and indicates very clearly that it has lost none of its malignancy by being transferred to America.

ENZOOTICS OF ERGOTISM.

Early in March, 1884, a disease among the cattle of Coffey County, Kansas, which was supposed by certain veterinarians to be foot-and-mouth disease in a most virulent form, was brought to the attention of the officers of that State; and such exaggerated accounts were sent to the press from day to day as to cause a feeling of insecurity and alarm among all engaged in the live-stock industry of the West. The 3d of March, Dr. Wilhite visited the farm of Mr. Daniel Keith, located in Coffey County, 4 miles northwest of Neosho Falls, and pronounced the trouble among his cattle to be foot-and-mouth disease. The same day Governor Glick telegraphed as follows:

TOPEKA, KANS., *March* 3, 1884.

HON. GEO. B. LORING,
Commissioner of Agriculture:

A very malignant disease has broken out among the cattle in Neosho County, this State. It is supposed to be the foot-and-mouth disease. The feet become sore and soon rot off. The disease sometimes extends to the knees. Over a hundred animals have been attacked in a few days. Great consternation among the cattle owners. Can your Department send here a competent veterinary surgeon? No one here can advise what to do.

G. W. GLICK, *Governor of Kansas.*

Two days later the following dispatch was received:

TOPEKA, KANS., *March* 5, 1884.

HON. GEO. B. LORING,
Commissioner of Agriculture:

Veterinary surgeon reports disease of which I advised you to be foot-and-mouth disease.

G. W. GLICK, *Governor of Kansas.*

On the receipt of this dispatch, and similar representations by the Senators from Kansas, Dr. M. R. Trumbower, a veterinarian whose previous reports of various diseases had led us to put great confidence in his knowledge and judgment, was directed to proceed at once to Neosho Falls, and make an early report in regard to the nature of the disease among cattle which was said to exist at that place. He started on the 6th, but could not reach the affected farms until the 9th of March.

In the mean time, at the request of the governor, General Augur detailed Dr. Holcombe, of the army veterinary service, to make an immediate investigation. In company with the governor, the secretary of the State Board of Agriculture, and a delegation of citizens from Emporia, Dr. Holcombe reached Neosho Falls March 6, and after a hurried examination of the Keith, Goodrich, and Beard herds he reported that the disease was the genuine epizootic aphtha of Europe. The following dispatch was received at the Department of Agriculture the same day:

NEOSHO FALLS, KANS., *March* 6.

HON. GEO. B. LORING,
Commissioner of Agriculture:

Veterinary surgeons A. A. Holcomb and A. H. Wilhite have to-day made an examination of the infected cattle, and pronounce it foot-and-mouth disease. Over 100 head are affected, but the disease is confined to stock cattle on a half dozen farms.

G. W. GLICK, *Governor of Kansas.*

When Dr. Trumbower reached Neosho Falls, he found an excited throng of people who urged upon him the necessity of making an immediate diagnosis, and relying rather upon the representations of others, which in many important respects proved to be incorrect, than upon what he was actually able to see, and a careful judgment based upon this alone, he was led to concur in the opinion of the professional gentlemen who had been upon the ground for the preceding three or four days.

On March 10, Dr. Holcombe made his formal report to the governor, in which occurred the following sentence:

> That it is foot-and-mouth disease cannot be doubted when the symptoms are considered; for to recapitulate, the various cases show vesicles and ulcers of the mouth; vesicles and ulcers in the cleft of the hoof; suppuration and sloughing at the foot; ulcers of the rectum; vesicles and ulcers of the udder; diarrhea; a temperature varying from 101 to 104.4 degrees Fahr., and the most remarkable emaciation even in cases where the appetite is good.

The excitement now became so great that by your direction I left Washington, March 13, to investigate the nature of the disease and to see what action, if any, was necessary to hold it in check. At Chicago I learned of what was supposed to be a similar outbreak at Effingham, Ill., and was requested by Dr. Rauch, secretary of the State Board of Health, and by others largely interested in the cattle industries of the State, to make an immediate investigation. The condition of affairs in Kansas, however, was so urgent that I concluded to press on as rapidly as possible.

I reached Topeka March 15, and immediately had an interview with the governor and with the secretary of the State Board of Agriculture. I was informed by both that the malady was undoubtedly foot-and-mouth disease, but that it was so quarantined that there was little danger of its immediate extension; and at the request of the governor I proceeded the following day to Pawnee County, to investigate a supposed outbreak of pleuro-pneumonia. I found the cattle there to be suffering from chronic indigestion, the result of feeding too exclusively for a long time on dried sorghum with a probably insufficient water supply.

I at once returned, reaching Neosho Falls March 19, and after a careful investigation was able to telegraph you on the 21st that the affection was not foot-and-mouth disease, but that it had been produced by local causes and that there was no danger of its spreading.

Returning through Topeka, I reported my conclusion to the governor and was informed that experiments would be made with susceptible animals to decide the nature of the disease. I was invited to assist in these experiments, and at first decided to do so, but I soon learned that the investigation necessary to satisfy Dr. Holcombe, who had just been appointed State veterinarian, was such as to require much more time than I could give to it. I accordingly visited Kirksville, Mo., by your direction, where I found an outbreak of the same disease as existed at Neosho Falls, and from there returned to Washington.

In Kansas I met Professor Stalker, of Iowa, Professor Faville, of Colorado, Colonel Groom, of Texas, and Dr. Hopkins, of Wyoming, who had been commissioned by their respective States to report on the nature of the disease and the necessity of quarantining all cattle, sheep, and pigs from Kansas. I was also requested by the secretary of the Illinois State Board of Health to inform him if there was any necessity for his State to adopt similar measures. Fortunately, each of these States received a report that foot-and-mouth disease did not exist in Kansas, and what threatened to be an almost complete suspension of the live-stock business of the West was averted. There is no doubt, however, that the cattle industry suffered a considerable loss from the excitement. The market became unsteady, the price of cattle declined, and buyers became exceedingly cautious.

April 9 I received information that Dr. McEachran, principal of the Montreal veterinary school and live-stock inspector for Canada, had visited Neosho Falls, Kans., and Effingham, Ill., as the representative of the Canadian Government, and positively asserted that the malady at both places was the real foot-and-mouth disease of Europe. April 10, a telegram from the State agent for Kansas of the United States Department of Agriculture conveyed the information that the State veterinarian had just reported to the governor that six healthy cattle cohabited with the sick animals had all contracted the disease, and that further experiments by inoculation would at once be made. A letter from the governor of the same date, received two days later, contained similar statements. A few days later still, an item appeared in the press dispatches from Washington, stating that "a private dispatch was received here to day from the governor of Kansas, saying that cases of sickness among cattle which had been most carefully examined had turned out to be true foot-and-mouth disease. He was afraid that some cases had got in the herds. There was an attempt made at first to keep the matter quiet, but the information was deemed such as should go to the public."

It now seemed that a repetition of the former excitement and panic was about to occur, and by your direction I visited Kansas a second time with instructions to make such experiments as might be necessary to demonstrate the non-contagious nature of the disease beyond question. I reached Emporia April 20, and was there met by a telegram from Neosho Falls asking me to join the State veterinarian and Professor Law at the governor's office on the morning of the 22d. Not intending to turn backward until the difference of opinion was conclusively settled, I telegraphed in reply requesting these gentlemen to meet me at Emporia on their way to Topeka. This they did on March 21, and I had a conference with them, at which the State veterinarian admitted that all attempts to convey the disease by inoculation upon cattle, rabbits, and sheep had failed; that the second experimental lot of cattle which had cohabited with the first lot when they were supposed to be suffering with foot-and-mouth disease had not been in the least affected; that the foot symptoms of the first lot had only been noticed with two animals, were very slight and of exceedingly short duration; and that, finally, whatever the disease might be, it was not the continental foot-and-mouth disease.

After receiving this information I returned to Topeka, attended the meeting of the live-stock commission in the governor's office, when the State verterinarian reported that the malady at Neosho Falls was not the foot-and-mouth disease, and the governor sent out a dispatch to the same effect.

On my way to Washington I visited the herds in the vicinity of Effingham, Ill., examined the cattle and the food and assured myself that the disease there was identical with that in Kansas and Missouri, and that it was in every case traceable to the ergot which existed in great abundance in the hay.

SITUATION OF THE AFFECTED HERDS AND BRIEF HISTORY OF THE DISEASE.

The cattle disease in Kansas which recently attracted so much attention from its supposed identity with the contagious foot-and-mouth disease of Europe, was first noticed in the herd of Daniel Keith about the 23d or 24th of December, 1883. Mr. Keith's farm is located 4 miles northwest of Neosho Falls. The first to sicken were some yearlings, which were noticed in the morning standing "humped up," with drooping heads and jerking the hind feet in a peculiar manner. These would walk but little and would soon lie down. Within two or three days they were inclined to lie continually. The feet were examined and found free from mud; the interdigital space was described as red, swollen, and sensitive, the toes spread apart. The feet began to swell at the coronet, or as high as the fetlock; a line of separation was established, and pus appeared within two or three days from the first symptoms. The mouths were not examined, but the animals were supposed to be eating all right.

On or about December 10, Mr. Keith had purchased 63 head of yearlings of Mr. Davis, all of which had been gathered within a radius of 10 miles. Two cows and 6 yearlings were bought of Alexander Linn, 1 mile down the river from Neosho Falls. This lot of yearlings were said to have sickened within a few days after their arrival on the Keith farm; it is believed that some were sick within three days and that all were suffering within a week, and during this time they had been fed on shelled corn and mowed oats. There appears to be some doubt as to how severely they were affected, whether they were all attacked on the same day, and the exact number of days they were on the farm before showing any symptoms. While it was asserted that they ate no hay it was admitted that there was probably some hay in the racks. Eight other animals were purchased about the same time of neighbors living within 2 or 3 miles.

By January 1 he had between 20 and 30 head sick, a number of new cases being observed each day. March 9 Dr. Trumbower found a red yearling steer with a very hot mouth, mucous membranes much reddened, a vesicle the size of a dime on the soft palate, and two smaller ones on the tongue. There was also a small ulcer on the mucous membrane of the rectum; the temperature was 104.4° F.; the animal was lying down, and when forced to rise it moved very stiffly, but there was no swelling of the feet. The following day the vesicles were found ruptured, and in their place was a deep, red cavity which bled when touched. Temperature still 104.4°. March 20 this animal appeared well.

The cattle on this farm were divided into two lots, which were in adjoining inclosures, and were separated only by rail fences. The second lot contained 40 two-year-old steers, purchased about November 1, and was free from disease until February 28, though some of these had broken through the fence at times and mingled with the diseased part of the herd. March 10 about 10 or 12 animals in the second lot were sick. Three days later Dr. Trumbower found 2 that would lose all four feet. At this time there were 118 head of cattle on the farm, of which

74 were more or less affected. Nine animals had one foot off, 4 had two feet off, 1 four-year-old cow lost both hind feet and a toe from one fore foot, 3 others were affected in but one foot, 6 in two feet, and 1 in three feet. In nearly all that showed lameness there were more or less mouth symptoms.

Across the road, and nearly opposite to Mr. Keith, lives Edward Hindman. It was here that the cattle belonging to A. C. Goodrich were located. The 10th of March this herd numbered 96 head. The first animal affected was a milch cow, noticed to be lame January 10. No other cases occurred until February 14 or 15, when one was seen to be lame in the morning after a heavy ice storm. The following morning 16 were lame. After that he discovered new cases almost daily, and on March 14, when they were separated from the well ones, 65 head were affected. March 19 the most severe cases were as follows: 18 animals had lost both hind feet, 5 had lost one hind foot, 1 had lost both hind feet and one fore foot, and 1 had lost all of its feet. Seven of the others were lame in the hind feet, and the remainder of the 65 head affected were more or less lame.

In each of the above-mentioned herds the sick animals at the time of my visits, March 19 and 22, had small erosions of the mucous membrane of the mouth, and 2 or 3 had hard yellowish crusts in the same situation an inch or more in extent. In no case were these sufficient to interfere with mastication. This lesion was more noticeable in the old chronic cases than in the recent ones.

The third herd which contained diseased animals was located about 2½ miles from those already mentioned. It belonged to J. W. Beard. This herd contained 70 head of animals, and 3 cows and 2 steers are all that have been affected. All had been running together until the disease was noticed, when the sick were separated from the well. The first symptoms were observed on the 17th of February, when a cow was seen to be lame. The second one to go lame was a cow bought of Mr. Keith and brought to the farm February 18—she became lame about the 22d or 29th, accounts differing between these dates. The third one, a steer, became affected March 1. The fourth was a cow observed to be sick March 1 or 2. It is reported that she slavered very profusely. Mr. Beard states that he examined her mouth and found it very red and the tongue covered with little pimples. At ten o'clock next morning she died. This animal had not been lame. The fifth one was taken about the same time. It was lame in one foot, became better, was affected in a second foot, and was entirely recovered from lameness by March 11. At this time Dr. Trumbower found several small sores and discolored spots in the mouth, and the temperature was 102.5 degrees, or very nearly the average of cattle in health. The highest temperature found by Dr. Trumbower was that of the cow purchased of Mr. Keith, which reached 102.8 degrees—a point too low to indicate fever with any certainty.

The fourth and only remaining herd in the neighborhood of Neosho Falls was that of Christian Pribbernow, whose farm is located on Owl Creek, 10 miles southeast of the town. There were on this farm 183 head of cattle, and but 16 have shown any signs of the disease. This herd was made up as follows: 54 yearlings, 24 two-year-old steers, 13 two-year-old heifers with calf, 15 three-year-old steers, and 77 cows and heifers. The yearlings had been put in a separate pen and fed on oats and corn-fodder—none of these were affected. Three apparently well animals were lassoed and examined; their mouths contained small erosions and discolorations of the mucous membranes. The temperature of one, thought to be slightly lame, was 101 degrees; that of another, apparently in perfect health, was 103 degrees. Six of the affected ones have either lost their feet or have them in such condition that they will surely separate from the legs, and 2 others have lost digital bones. None of these animals have shown salivation or loss of appetite; but the mouths contained erosions and discolorations similar to those seen in other herds.

At Hall's Summit, a distance of 20 or 25 miles north from Neosho Falls, George R. Smith owned 2 cows. About February 1 one became lame; there was noticeable slavering and loss of appetite for several days. This cow calved February 29, and Dr. Trumbower saw her March 17, when the calf appeared well but small. The cow was reduced to a skeleton. Her right hind leg had broken off half way between the fetlock and hock joints, carrying with it the lower half of the metatarsal bone. The left hind leg was separating at about the same point. One toe of the left fore foot was coming off at the first joint.

Near Hartford, some 20 miles northwest of Neosho Falls and 15 miles west of Hall's Summit, was the farm of Mr. O'Toole, where another outbreak of disease occurred, showing precisely the same symptoms. The animals at this place were reported to have been killed before my visit, and consequently I did not see the herd. Dr. Wilhite, as I was informed, thought the first cases appeared about January 10. The first animals attacked were yearlings. Soon after all the calves became affected in the same way. Then the large steers in the feeding pen were attacked.

About the middle of March the governor of Kansas sent a veterinarian to investigate a disease which was reported to exist in Osborne County. According to verbal information which I received from the State officers while at Topeka, this disease was substantially the same as that which existed at Neosho Falls. The distance between these two points must be at least 175 miles in a direct line.

After investigating the disease in the neighborhood of Neosho Falls, I proceeded as directed to Kirksville, Adair County, Missouri, to visit herds at that place reported to be affected with foot-and-mouth disease. On March 27 I was at the farm of William Bragg, who lived 5 miles south of Kirksville. The disease in this section was first noticed here, but later 6 other herds, within a radius of 4 miles, have had affected animals. The only new animal introduced on the Bragg farm was a steer bought in the neighborhood about December 20. This was one of the first to sicken, but there was no disease on the farm from which it came. A cow that had been purchased a month earlier sickened about the same time. This was in the latter part of January. The weather had been extremely cold early in January, and reached 10° or 12° below zero at other times during the month.

At the time of my first visit there were 4 animals lying in the stable. One cow had lost a hind leg from about half way between the hock and fetlock joints; the bones had separated at the latter joint and the metatarsal bone protruded half its length beyond the flesh. The other hind leg was dividing at the fetlock joint. Six inches at the end of the tail was gangrenous, and was being separated from the remainder of the organ. There were a number of abrasions and small discolored spots in the mouth. A second cow had a healthy mouth; both hind feet were lost at the coronet, and the tip of the tail was gangrenous. A steer, probably two years old, had lost both hind feet at the fetlock, about an inch of the tail was lifeless, and the mouth contained a number of sores and discolorations. A second steer was in almost precisely the same condition. A third steer was walking around the yard, very lame, and had a large slough of the tissues on the posterior surface of the fetlock joint. A fourth steer in the pasture had both limbs as high as and including the fetlock joint stiff and cold. Still another animal was lame in the hind limb. Seven herds within a radius of 4 miles had suffered. Six abortions were reported.

April 24 and 25 I visited a number of the diseased herds in Effingham and adjoining counties in Illinois. The farm of Lemuel Faunce is situated 10 miles northeast of Effingham and one and one-half miles from Montrose. The first cases appeared in the latter part of December, and began with diarrhea and other signs of digestive disturbance. There were 21 head of cattle on the farm and no new ones had been purchased at the time of or immediately preceding the outbreak. Two cows, each of which had both hind legs affected, had been killed before my visit; 1 steer has a hind limb off at the fetlock; another has a clear line of demarcation formed at the fetlock, the part below being gangrenous; a bull has lost both toes from one foot and one toe from the other; 2 other animals were very stiff. One steer had two attacks and another had three attacks of lameness, and the latter entirely recovered. The animal that was first to suffer still had sores, *i. e.*, erosions of the mucous membrane on the upper lip and gums exactly like those which I saw when examining the affected cattle at Neosho Falls and at Kirksville, though four months had elapsed since the appearance of the disease. Some of the sick ones had slavered and smacked the lips, showing that the mouth was quite severely affected.

The horses on this farm had also been troubled with an eruption in the mouths which had caused salivation and loud smacking of the tongue and lips. These were now entirely recovered, though slight evidences of the sores on the lips were still visible. The horses were seen to have lost appetite in January or early in February. The last of February sores were observed in the mouths, and it was six weeks before these healed. Only one hog was kept, though many of the neighbors' hogs had been continually running around the pastures. None of these had been affected.

Three miles north and 1 mile west of Mr. Faunce's farm, Mr. Dubroc had yearlings in a high, dry lot, in which was an out-house for shelter, parly filled with hay. All of these were affected and all recovered. There were here 160 head of cattle, only 8 or 10 of which, all told, were lame. Ten or 12 goats were running with the cattle, but remained well; the two places mentioned above were so situated on different roads that there was little if any passing from one to the other, and the outbreaks were therefore independent of each other.

Other cases of the disease occurred on the farm of Mr. John Mason, who lives near Wheeler in Jasper County. This gentleman owned 120 head of cattle, of which 17 had been affected. Six animals were so bad that they had been killed; 2 others remained, one of which had lost a foot, and the second one would lose both of the posterior feet at or above the fetlock; a part of the tail of this one was also gangrenous. Nine others had been more or less lame but had lost no limbs. On this farm and in close proximity to the cattle were 25 horses and mules, 100 hogs and 40 sheep, all of which had been free from disease.

In the town of Wheeler, a single family cow was found with the lower parts of the posterior limbs separating as a result of dry gangrene. This seemed to be the only sick animal in the town.

Mr. Keating, who lives 6 or 8 miles from Effingham, had also suffered from the same disease. His herd consisted of 45 young cattle and 6 cows. None of the cows were affected, and it is worthy of remark here that they had been fed upon hay harvested in 1882. The young cattle were fed upon the crop of 1883, and of these 8, which were in a very bad condition from the loss of their limbs, had been killed; two others were still alive with the feet off at the fetlock. About half of the 45 young cattle were more or less affected. There were 60 sheep and a number of hogs on this farm, none of which had shown any signs of disease. The cattle here were attacked about the 8th of January.

These farms are mentioned as examples of what had occurred at seventeen or eighteen different places that I learned of within a radius of 15 miles from Effingham. As a matter of great interest connected with this subject, I was informed by a number of people that there had been a greater number of abortions among mares, and more cases of difficult parturition during the past winter and spring than was ever known before.

Other herds were reported on good authority to be affected in the same manner at different points in Missouri, Illinois, Iowa, and Colorado.

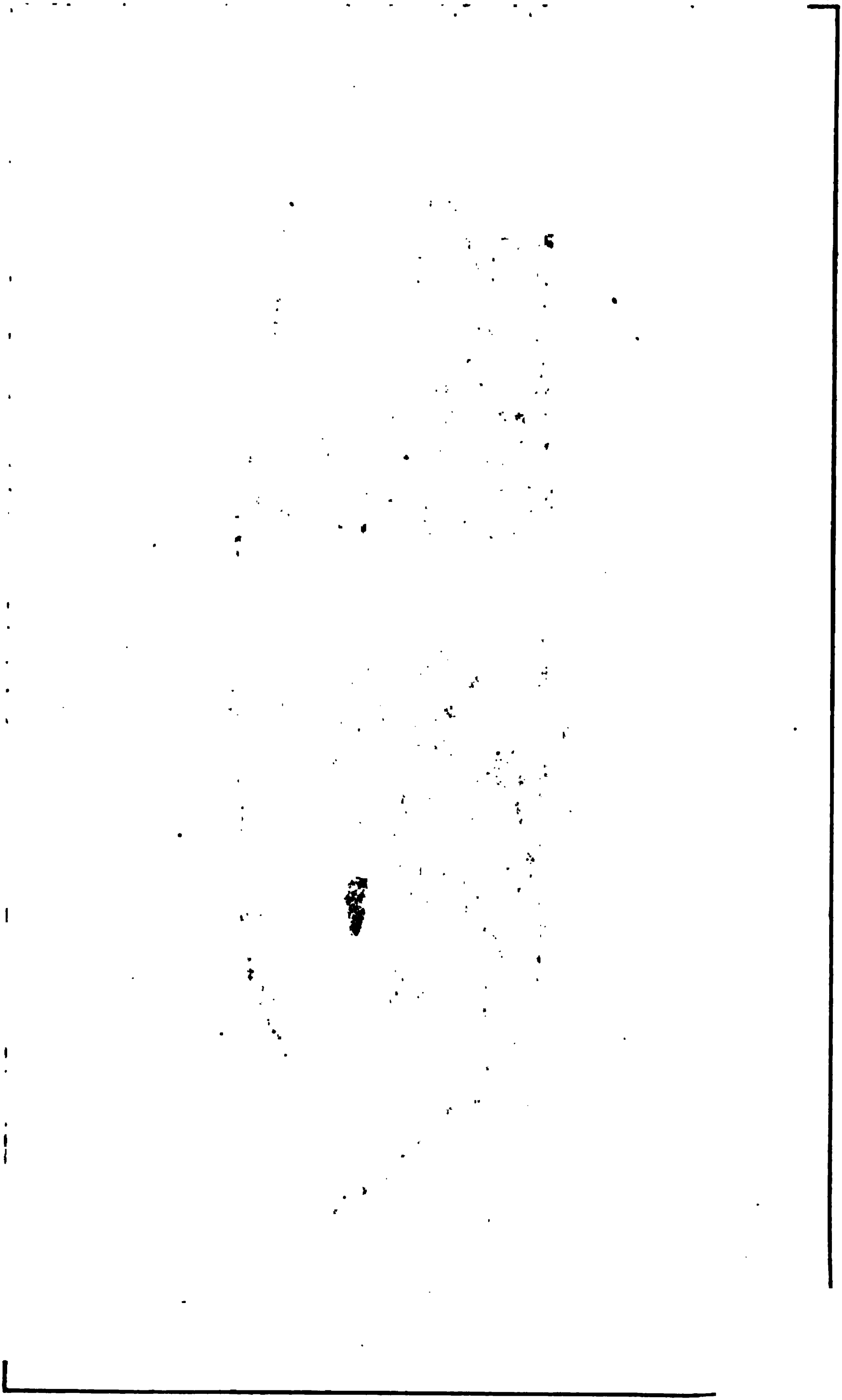
CLASS, CONDITION, SURROUNDINGS, AND CARE OF THE ANIMALS.

All the diseased animals on the farms visited by me were stock cattle in medium to thin condition. Those worst affected, in which one or more limbs were separating as a consequence of dry gangrene, had evidently lost much flesh during the progress of the disease. There were no fat cattle on any of these farms. At Mr. O'Toole's it was said by those who visited the place the fattening cattle were attacked as well as the stock cattle and calves.

On most of the farms there were cattle of all ages—calves, yearlings, two-year-olds, three-year-olds, and cows. The calves and yearlings seemed to escape in a greater degree than the older cattle. In the Goodrich herd were 20 calves which occupied a lot through which the other animals were frequently driven to water and into which some of the lame ones were placed. This lot was separated by an open fence from that in which were kept the worst diseased animals of the herd, and yet not one of the calves suffered in the least. At Pribbernow's were 54 yearlings running with the other cattle, and from which the worst affected ones were only separated by a rail fence, and all of these escaped. At Keith's were 2 young calves sucking diseased mothers, but themselves in good health. Here also were hogs and a litter of young pigs running in the same lot with the sick cattle, but free from any signs of disease. At Kirksville sheep had been running with the cattle and were also healthy. In Illinois, sheep, swine, and goats mingled with the affected herds with perfect safety.

The winter has undoubtedly been a severe one upon the stock of the Western States, and the cattle were consequently somewhat below the average condition at this season of the year. The appearance of the disease cannot be explained by this fact, however, since thousands of healthy herds were in worse condition than those on the farms in question. Some of these herds, and noticeably that of Goodrich, were in much better than average condition; they had evidently been well fed and cared for.

There was nothing in the surroundings of the affected animals which would explain the development of the disease. The feeding lots in most cases were unusually dry and the disease had appeared at a time when all mud was frozen solid. The soil did not contain enough alkali, V



ERGOTISM

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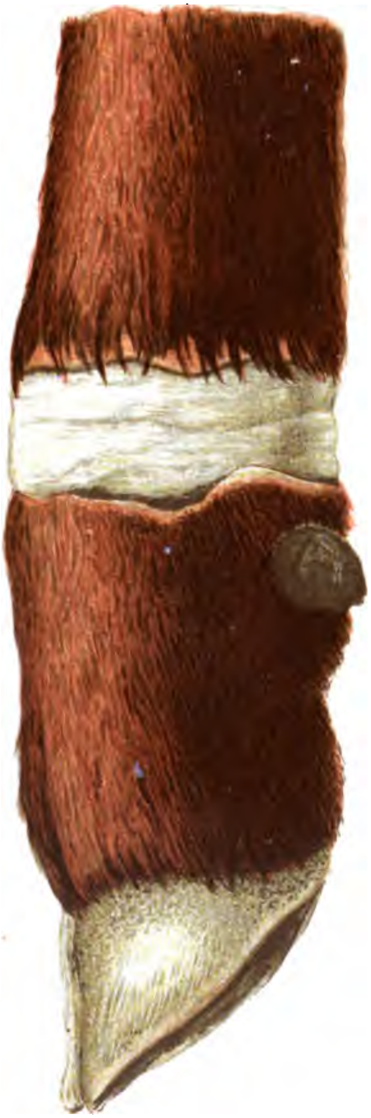
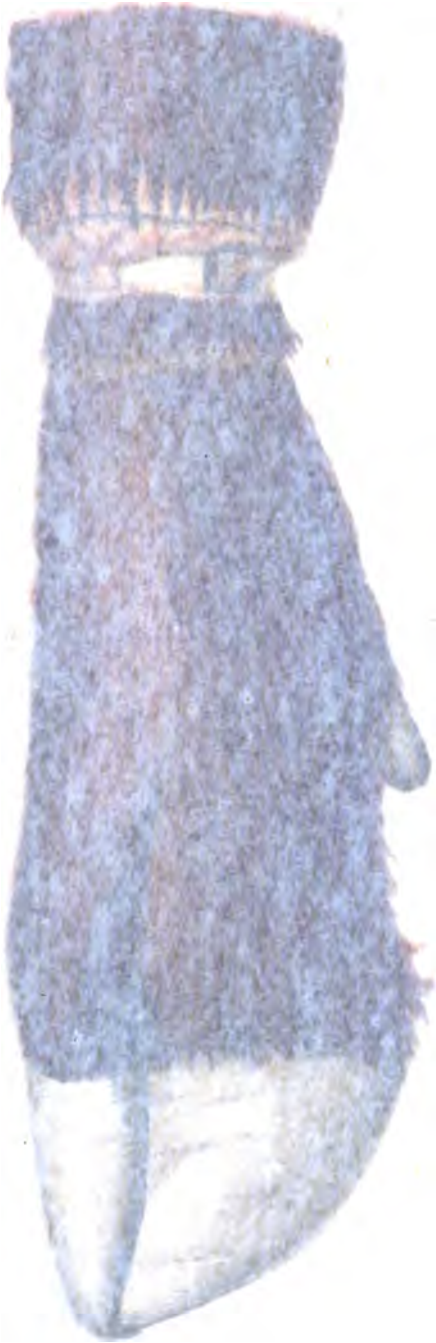
Plate V

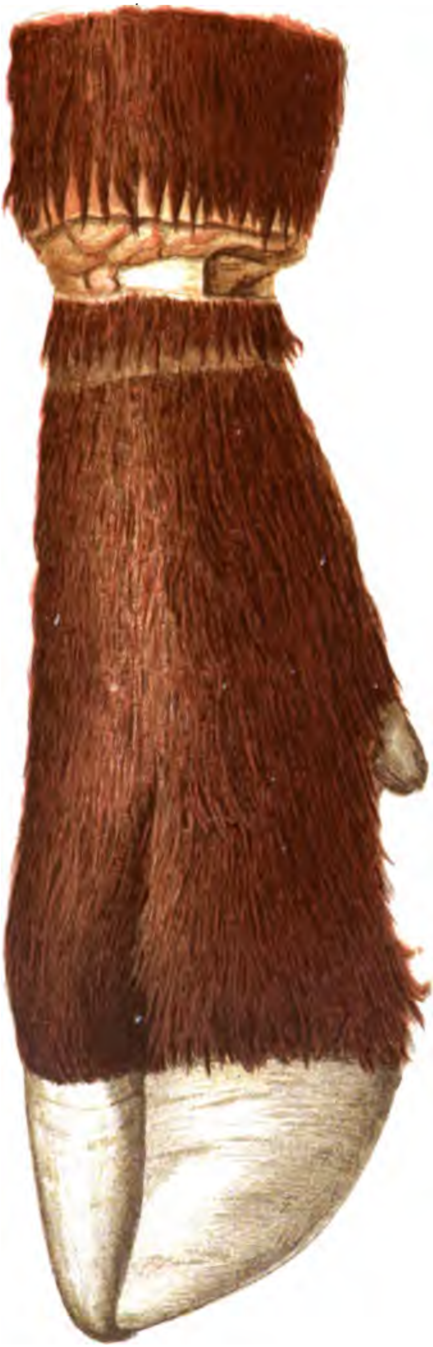


ERGOTISM

Plate VI



ERGOTISM (KANSAS)



ERGOTISM (KANSAS)

UNIV. OF MICH.

even at Neosho Falls, to make it at all probable that this could have been the exciting cause of the disease. As is usual in the management of cattle at the West, the herds were without shelter. At Keith's the cattle lots were in a ravine protected by timber; on some of the farms there was little protection of any kind. Such a condition, however, is so common that it could not be regarded as having much influence in the production of this trouble. All of the affected herds seem to have received ordinarily good care. Keith had fed some of his cattle shelled corn and mowed oats in addition to hay. Pribbernow had fed his yearlings on millet, oats, and corn-fodder, and consequently they had eaten less hay. Beard had fed shocked corn. In Illinois, Keating had fed liberally on corn. On most of the farms the water was very good, but probably deficient during the cold weather. Keith had pumped water from his well; Goodrich's cattle drank from a pond; Beard's from the river, and Pribbernow's from a creek. It was necessary to cut holes through the ice and these would soon freeze over; consequently, it may be admitted that in most cases there might have been a deficiency of water.

When the animals first became lame it was supposed that mud had collected between the toes, and, becoming hard, was producing irritation. The animals were caught and their feet cleaned, but this had no effect on the development of the disease. It is evident that the animals were cared for as well as is ordinarily the case in this section of the country, and that the slight deficiency of water and the exposure to cold were accessory rather than the exciting cause of the disease.

SYMPTOMS AND CHARACTERS OF THE DISEASE.

The first symptoms of disease in the Illinois epizootic were diarrhea, lameness, stiffness of the lower joints of the affected limb, and coldness and insensibility of the same parts. In Kansas this derangement of the digestive apparatus was not noticed. At all the places visited, however, the lesions of the feet were of a common character and were produced by a common process. In the more severe cases a constricted band formed around the limb at the point separating the gangrenous from the living flesh. So marked was this constriction that some of the owners looked upon it as the initial lesion of the disease and cut across it with a knife in the hopes of re-establishing the circulation. It is needless to say that this hope was delusive, since the part below the constriction was entirely lifeless before this was formed. The constriction was the first step in the effort of nature to rid the body of parts that were of no further use to it.

The next step in the process of separation was a crack in the skin at the upper edge of the band of constriction, which gradually extended toward the center of the limb, the softer parts dividing first and the tendons and ligaments resisting much longer. Generally this separation was in the vicinity of a joint, and in this case, as the lower members of the limb were lost a comparatively even surface was left which healed readily. Some animals lost only a toe, the dividing line passing through the joint between the *os pedis* and *os coronæ;* others lost both the *os pedis* and *os coronæ;* still others lost the three lower bones, and the line of separation passed through the fetlock joint, while in the most severe cases the line of constriction formed at the upper third of the metatarsal bone and the fleshy parts sloughed off, leaving the uncovered bone protruding for more than half its length. Plates V and VI are drawings made from limbs which I secured in Kansas.

It was reported by some of the veterinarians that small vesicles were formed in the interdigital space and about the coronet, and this was doubtless true, as such vesicles are not uncommon in gangrene; but their appearance was far from being the rule, as I did not succeed in finding a single one in all the animals that I examined. In nearly all of the cases, whether the foot was affected with dry gangrene or whether there had been simply lameness without death of the part, the skin of the interdigital space and about the coronet was perfectly preserved. There was loss of neither epidermis nor hair, as there certainly would have been had the disease commenced by a superficial inflammation in this region and extended to deeper parts of the foot or to higher parts of the limb. Indeed there were no abscesses, no burrowing of pus, no ulceration about the feet, which could lead one for a moment to suppose that the cause of the disease had commenced its action externally and extended gradually to the interior of the limb. On the other hand, the fact that the skin was intact in the great majority of cases, that the part was cold and insensible almost from the first, and that the line of separation passed entirely through the limb, removing one or more phalanges as completely as it could have been done with a knife, was sufficient evidence that the disease had an internal origin.

The gangrene was not confined to the feet, however, for in Kansas, Missouri, and Illinois there were individual animals which were losing from 2 to 6 inches of the lower part of the tail by exactly the same process. The portion below the dividing line was very dry and hard, while the line itself was sharply defined, as though it had been a knife-cut. With the greater part of the animals affected in the feet a careful examination of the end of the tail revealed a slough of greater or less extent; sometimes it was simply the skin at the tip that was affected, but oftener one-half inch, 1 inch or 2 inches would be found discolored, lifeless, and dry. In a very few cases a part of the ear was found in the same condition.

One of the most interesting features of the enzootic, because it had not been heretofore described, was the implication of the mucous membrane of the mouth. With some animals this was limited to a more or less diffuse red discoloration, without loss of substance. More frequently there were circumscribed dark red spots or patches, from a fourth of an inch to an inch in diameter. Very often there was loss of substance—erosions from a third to a half inch in diameter. Some of the veterinarians reported that they had discovered blisters in the mouths, and it is not unlikely that these erosions in their first stages were more or less vesicular in character, but I was not fortunate enough to see them in this stage at any of the places visited. In some animals the part of the membrane that was being lost was still attached by shreds, in others it was entirely removed, but in no case did I see anything of the nature of a vesicle. None of the erosions presented the appearance of ulcers, or showed any considerable inflammation. They were dark-colored, the borders were not elevated, and the surrounding blood-vessels were neither prominent nor injected. It appeared to be only the superficial layer of the membrane that was involved.

In a very few animals a lesion of a different character was observed in the mouth. In these cases an irregular patch of mucous membrane from 1 to 2 inches in diameter was elevated, corrugated upon its surface, hard, insensible, and of a light color, tinged with pink and yellow. It seemed to be a circumscribed gangrene of the mucous membrane, the dead parts being partially decolorized by soaking in the fluids of the mouth.

There was also an evident irritation of the mucous membrane of the posterior parts of the alimentary canal and organs of generation. That covering the rectum and vagina was generally red, covered with mucus, and presented spots denuded of the epithelium. In Missouri six cases of abortion in cows were reported, and in Illinois there were many cases of abortion and difficult parturition with mares.

The constitutional symptoms were not very marked. The temperature of the animals which I examined was about normal, with the exception of a few from which one or more limbs were sloughing and with which there was suspicion of septic poisoning. Drs. Holcombe and Trumbower observed high temperatures (104 to 104.8 degrees) in some cases in the early stages of the affection.

In those animals which recovered after showing lameness there was no loss of substance or inflammation of the skin as would have resulted from freezing to a sufficient depth to cause lameness. In these animals the lameness and stiffness of the lower joints were the only symptoms of the disease in the feet, though the same animals frequently showed erosions in the mouths.

EVIDENCE POINTING TO ERGOT AS THE CAUSE.

In each of the herds which I visited, with the single exception of Beard's, there were typical cases of dry gangrene of the extremities, with an evident preference for the posterior limbs. In the most severe cases there was complete death of the leg as high as the middle portion of the metatarsal bone. This dead part was sharply defined, first by a constriction and later by a crack from the living flesh above. It was not a death of the superficial structures alone, but the skin, tendons, and bone were all involved, and every part of the leg below the line of separation just referred to was completely lifeless. A study of these legs showed very clearly that the disease had not begun at the hoof or in the interdigital space and progressed upward, for these parts had not been changed by disease of any kind previous to the death of the whole affected part, which had evidently occurred very suddenly. To my mind this condition made it very plain that the trouble was not the result of any disease which had begun in the interdigital space, or in the skin around the coronet. There could be no mistaking the fact that the worst affected animals presented typical cases of dry gangrene, and the problem to be solved was to determine which of the conditions that these animals were subjected to would satisfactorily account for the enzootic. When we turn to veterinary literature for information in regard to the accepted causes of dry gangrene, we learn that there are very few agencies which are liable to affect a number of animals at a time and are capable of producing this effect. Compression, burning, caustics, plugging of blood-vessels, and ergot about completes the list of those that would be at all likely to produce dry gangrene in young animals, and of these the last is the only one that could have possibly been instrumental in developing the outbreaks in the West.

The peculiarities of the disease led me to examine the feed to learn if any unusual quantity of ergot could be found. The result of this examination was to show that at every one of the farms where the diseased cattle were located, hay had been fed which contained one or more grasses ergotized to an extreme degree. At Keith's, Beard's, and Pribbernow's, in Kansas, there was a large proportion of wild rye (*Elymus virginicus*, variety *submuticus*) which contained an extraordinary quantity of ergot. In many heads half the grains and in other heads every grain had been replaced by the fungus. Careful weighings of heads brought to Washington, and from which some of the ergot had been lost in transit, gave in one case 12 per cent., and in another case 10 per cent., as the proportion of ergot. Now, if the head represented one-half the weight of the entire plant, from 5 to 6 per cent. of the weight of the rye must have been ergot; and if one-fifth of the weight of the hay was made up of wild rye, then a 20-pound ration of hay would contain about 4 ounces of ergot.

As is always the case where an attempt is made to account for results when the conditions affecting these have not been intelligently observed and carefully recorded at the time, we found some apparent discrepancies in the ergot theory. The greater part of these have been explained in a remarkably satisfactory manner, and if we could know every circumstance connected with the feeding and care of the animals for thirty or forty days preceding their illness, doubtless the most critical could be satisfied as to the cause of the disease in every subject. As we are compelled, however, to rely upon the more or less defective memories of the owners of the cattle, who, of course, did not make their observations in the light of subsequent developments, we must accept the situation as we find it and consider ourselves fortunate if a connection can be traced between cause and effect in the greater part of the cases. An exact estimate could not be made of the quantity of ergot in a given quantity of the hay in Kansas, but the weight of ergot in the heads of wild rye indicated this very closely. The head shown in Plate VII, Figure 3, is a good representation of this plant as it existed in the hay.

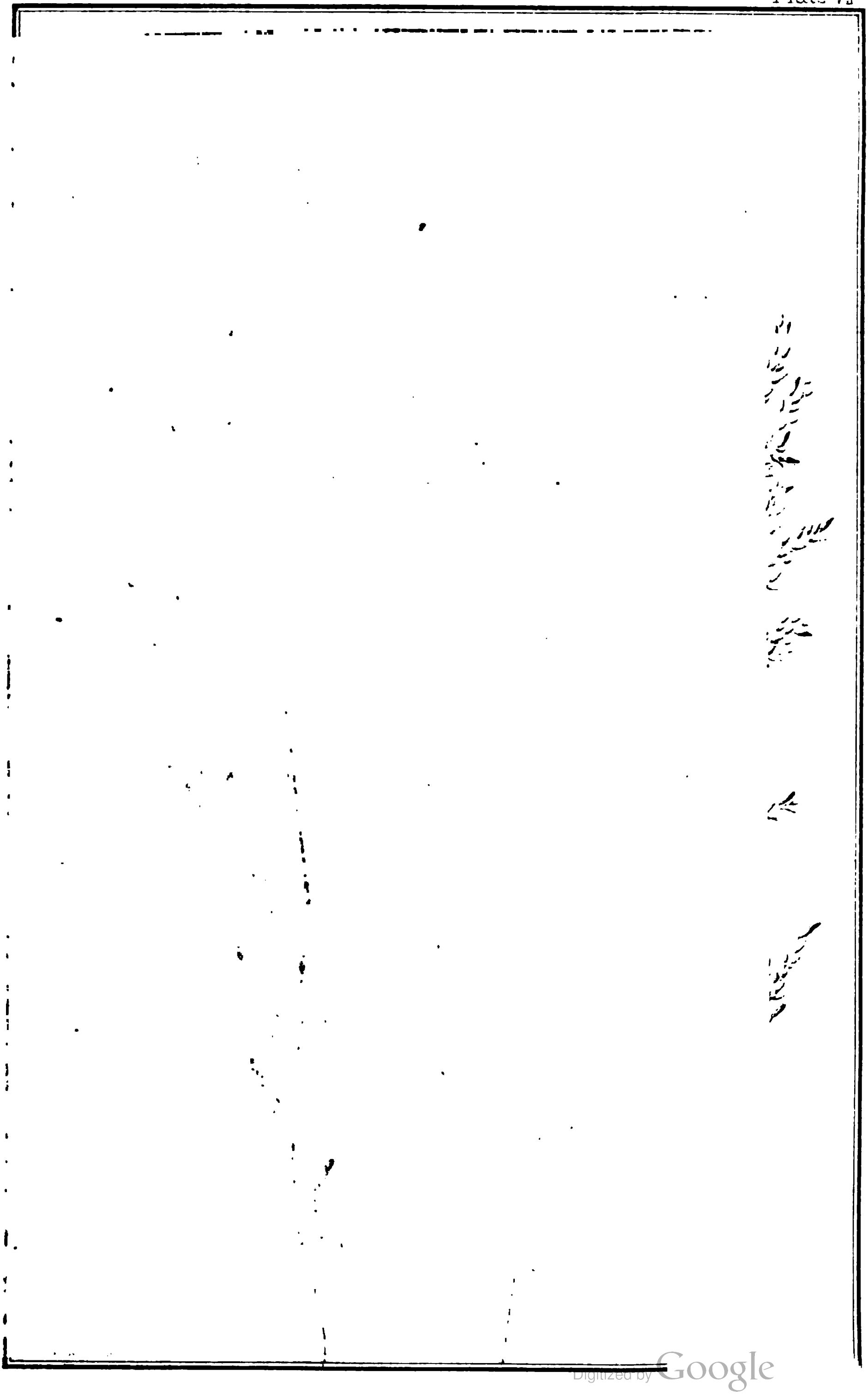
In Missouri the hay was made up mostly of red top (*Agrostis vulgaris*), but also contained some blue grass and timothy. The red top and blue grass contained a very large proportion of ergoted grains, and an occasional head of timothy was also affected. Figures 1, 2, and 4, Plate VII, are drawings from specimens of these grasses taken from the hay-racks at which the diseased cattle were eating.

In Illinois the hay was almost entirely composed of red top, and this contained a relatively large amount of ergot. Careful weighings of specimens of this hay and the ergot which it contained, from two of the worst affected farms, demonstrate that every 75 pounds of hay contains 1 pound of ergot; or, in other words, an animal eating 20 pounds daily of this hay consumed 4.2 ounces of ergot. Doubtless this quantity might be taken daily for a considerable time without producing appreciable effects under some conditions, but when the circulation in the extremities is diminished by extremely cold weather, and when in addition to this the water supply is limited, then ergot in this dose, continued day after day, becomes very dangerous.

In Kansas I examined the hay on adjoining farms where no disease had appeared, and I found a very much smaller proportion of ergot. At the Dibble farm, which joins Keith's, one might examine a dozen heads of rye without finding a grain of ergot, and the same was true of hay found in the town of Neosho Falls. In Illinois, at two farms, I saw hay of the crop of 1882 and also that of 1883, and while the former contained some ergot the latter contained a greatly increased proportion. It had been noticed by the people here that the red-top hay of the crop of 1883, for some unexplained reason, was greatly inferior; that animals neither relished it nor thrived when fed upon it, and it sold for $3 a ton when other hay would bring $10. At Keating's the animals fed on the hay of 1882 escaped the disease entirely, while those fed upon the hay harvested in 1883 alone suffered.

Evidently the year 1883 was a favorable one for the production of

Plate VII



ERGOT IN HAY.

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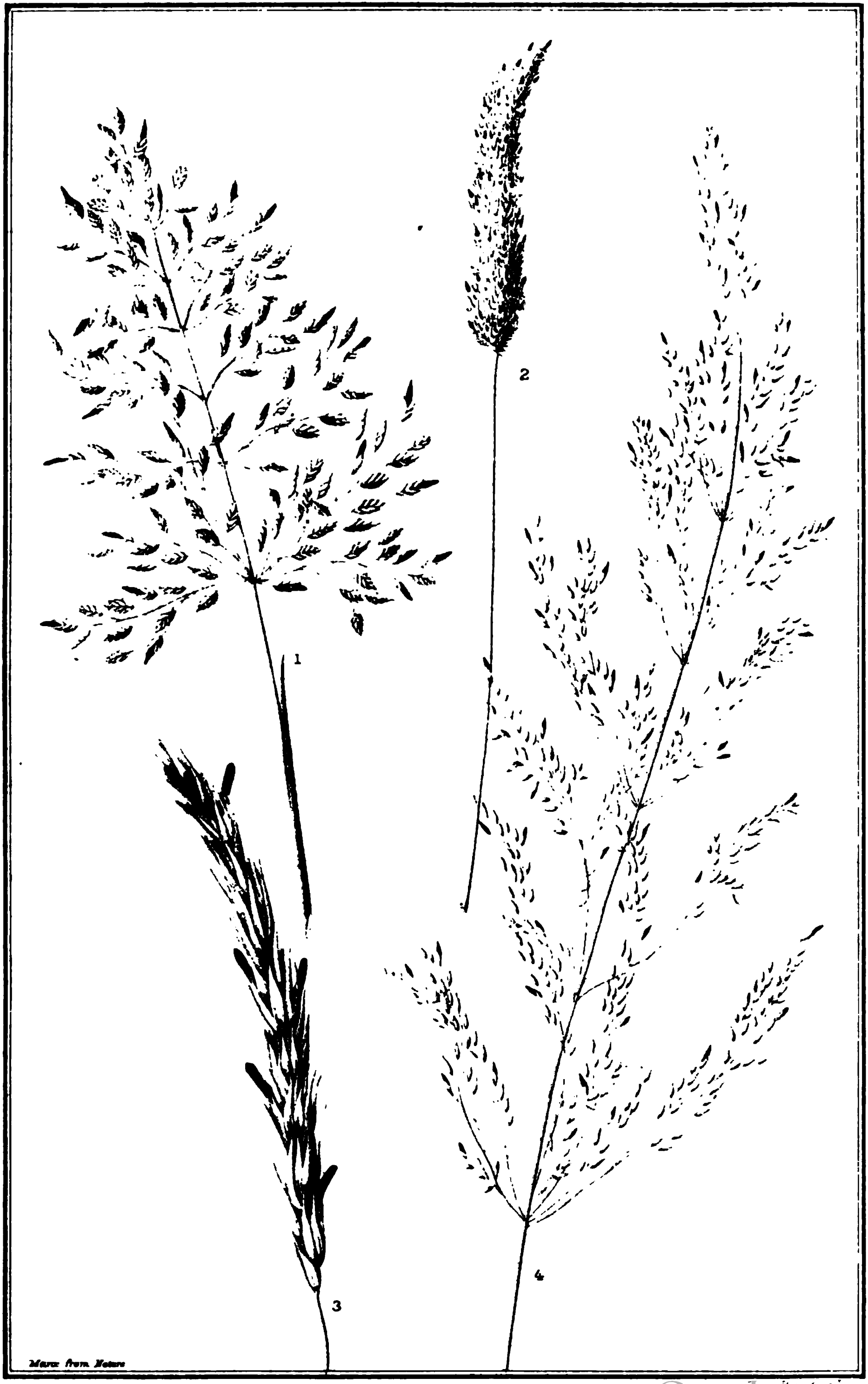

Marx from Nature

Chas Pier & Co.

ERGOT IN HAY

ergot over a very large area of the Western States, but the local conditions of soil and situation and the time of cutting the hay had a very great influence on its development. All of the ergoted hay of the affected farms in Kansas was cut from bottom lands, and in Missouri and Illinois it was grown on very level prairies the drainage of which was very imperfect. Again, the early cut hay was comparatively free, when that allowed to ripen was badly affected.

In brief, then, our reasons for considering the disease to be ergotism were, first, the character of the lesions, which were such as have always been ascribed to ergotism in the past, and as could scarcely be produced in so many animals from any other known cause; and, secondly, the extraordinary proportion of ergot found in the food of the animals on every affected farm.

It is very probable that the cold weather had a considerable influence in developing the effects of the ergot, and the greater part of the cases were first noticed during or soon after such weather. Many cases occurred soon after a severe ice storm or sleet. Again, with the appearance of milder weather new cases ceased to appear, although the same hay was still being fed. The two or three new cases in Missouri were the only exceptions to this statement.

I have no doubt, therefore, that the cases which I investigated, and the similar cases which occurred about the same time in other localities, were cases of ergotism. Professor Law, of Cornell University, Professor Stalker, of the Iowa Agricultural College, and Professor Faville, of the Colorado Agricultural College, have seen similar cases in their respective States, and concur in the opinion that they are due to poisoning from ergot.

CHARACTERS WHICH DISTINGUISH THIS DISEASE FROM EPIZOOTIC APHTHA, OR FOOT-AND-MOUTH DISEASE.

History.—The foot-and-mouth disease of Europe is a specific fever which only arises by contagion from other affected animals. In the whole history of America there have been no spontaneous outbreaks of this disease, and in Europe the conviction is growing stronger every year that it has no other cause than contagion. We may accept it, therefore, as a fact that foot-and-mouth disease cannot occur in the United States except by the introduction of virus from abroad.

When a disease having some resemblance in its symptoms to foot-and-mouth disease is found in the interior of our country, more than a thousand miles from the ports where the contagion must necessarily be introduced, it becomes a matter worthy of the most careful consideration to determine if there was any means by which this contagion could have been transported to the affected herd. When a contagious disease is spread broadcast over a country it may be difficult or impossible to trace many outbreaks; not so, however, with a single outbreak produced by so virulent a contagion as that of the disease under consideration. In such a case it would be remarkable if it could not be traced.

In the present instance the animals of the affected herds had been purchased or raised in the neighborhood; no foreign animals or people had been upon the farm where the first attacks occurred. Foreign cattle had for a long time been quarantined at the sea-board a sufficient time to make it impossible that this disease could have been carried by them to the West. It was absolutely impossible to find any satisfactory manner by which a foreign contagion could have been introduced.

This important indication seems to have been greatly neglected in deciding upon the nature of the disease in Kansas. It was said if this is foot-and-mouth disease we must acknowledge that we have it, whether we can trace its introduction or not. Plausible as this reasoning may seem we must admit that it is not always an easy matter to diagnose a disease off-hand from its superficial characters. And in the diagnosis of contagious diseases we must remember that the symptoms are but the expression of the effects of the virus, and that these symptoms may be simulated more or less closely by other agencies acting upon the animal economy.

The history of the origin of any disease believed to be contagious is, then, a most important part of the evidence to be taken into consideration before a diagnosis is reached. We may take contagious pleuro-pneumonia for example. Many cases of this disease resemble so closely spontaneous inflammations of the respiratory organs in cattle that it is absolutely necessary before a diagnosis can be reached to inquire if the contagious pleuro-pneumonia has been introduced or if the malady occurred spontaneously. The same principle holds good to a greater or less extent with other diseases, and it may be safely asserted that when the history does not receive proper consideration many mistakes will be made that otherwise might be avoided.

Contagiousness.—The virus of foot-and-mouth disease is one of the most active contagions known. The period which elapses between exposure and the appearance of the first symptoms of the disease is, as a rule, but two or three days; a very large proportion of exposed animals become diseased, and the plague spreads rapidly from farm to farm. As a result of these characters, within a week after the introduction of foot-and-mouth disease into a herd nearly every animal in that herd shows unmistakable evidences of having contracted it. A very small proportion of the animals may resist the contagion, but this proportion is much less than with most other contagious diseases, and is so small that it does not affect the rule just mentioned.

The disease at Neosho Falls showed very different characters from this. Goodrich's herd suffered in the largest proportion, 65 out of 96, or 63 per cent., being more or less affected. The first case here occurred January 10, and no others until February 15, or more than a month later. After this new cases continued to develop for two or three weeks. But in a lot adjoining that in which the sick cattle were placed there were 20 calves, which remained entirely free from disease. The isolation of these calves was not sufficient to hold foot-and-mouth disease in check for a single day; it was even said that the sick cattle had been driven through the calf lot to water, and that some of the smaller ones, when attacked, were placed with the calves.

At Keith's 74 out of a total of 118, or 63 per cent., were affected. At the end of the first week but 20 or 30 head had been attacked, and from this time new cases continued to appear until March, or during a period of two months. Here also it is to be remembered that in a lot of animals separated from the sick ones by a simple rail fence there was no appearance of disease until two months after it had attacked the first lot. Hogs were running in the lot with the worst cattle; they even ate the blood of the slaughtered ones and nibbled at the affected feet, but they did not suffer in the least. A sow had brought forth a litter of pigs in a shed which forms a part of the inclosure, and these were doing well. Two calves were sucking mothers under the influence of the disease but were themselves in good health.

At Pribbernow's only 8 per cent. of the animals had been attacked, and among a lot of 54 yearlings running with the other cattle there was not one case of disease.

At Beard's, in a herd of 75, the first animal was lame a week before the second was affected; and then another week passed before the others showed any symptoms. Here only 6 per cent. of the cattle on the farm were attacked, and one died within twenty hours from the appearance of the first symptoms.

At Kirksville the proportion of animals that suffered was not definitely ascertained, but there was no evidence of contagion, and sheep running with the affected cattle remained healthy.

In Illinois, on the Faunce farm, the horses suffered from an eruption in the mouth. The exact nature of this disease it was impossible to ascertain at the time of my visit. It may be remarked, however, that horses seldom suffer from foot-and-mouth disease; and that this is the only case which came under my notice on any of the affected farms where any other animals than cattle showed symptoms that were even suspected to be in any way connected with the disease among the cattle. In this instance the eruption in the horses' mouths could not have resembled foot-and-mouth disease very closely, for it remained at least six weeks, or three times the period of the latter disease. Here the neighbors' hogs which were running around the farm failed to contract any disease or to carry it to other farms.

At Mr. Mason's there was still more striking evidence to show that the disease was very different from epizootic aphtha. One hundred hogs and 40 sheep had been exposed, and not one suffered. Only 17 bovine animals out of 120, or about 15 per cent., showed any signs of the disease. At Keating's, 60 sheep and a number of hogs were exposed but all remained well. At Dubroc's, goats were exposed without suffering.

The disease, therefore, did not resemble foot-and-mouth disease either in the proportion of the animals attacked or its rate of extension, or in attacking other species of animals than cattle.

Occurrence at the same time on widely separated farms.—If foot-and-mouth disease had been introduced into the heart of the country in any of the extraordinary ways which were offered to explain its appearance, we surely cannot conceive of its being brought to so many widely separated points at about the same time, especially where there had been no communication between these places. In Kansas there were the Keith, Goodrich, and Beard herds which might be grouped together; 14 miles from these was the Pribbernow herd; 20 miles from any of these was the cow at Hall's Summit; across another space of 15 miles was the O'Toole herd; then it was necessary to travel nearly 200 miles to reach the Osborne County cases. Again, the same disease undoubtedly existed at several points in Iowa, Illinois, and Missouri. There had been no communication between these places, and if we assumed that they were the result of a foreign contagion it was necessary to conclude that a considerable number of independent introductions of this had occurred at very nearly the same time. This assumption, in view of the difficulties in the way of introducing a contagion to the interior of the country, and the impossibility of tracing such introduction at this time, was so improbable that it could scarcely be admitted even if all other evidence had pointed to foot-and-mouth disease.

Comparison of symptoms.—The symptoms of foot-and-mouth disease are constitutional and local. The constitutional symptoms are loss of appetite, elevation of temperature, and other signs common to fevers. The local symptoms consist in an eruption of blisters in the mouth, between the toes, about the coronet, and on the udder and teats. In order to understand the difference in symptoms between the recent disease in the Western States and foot-and-mouth disease, it is necessary to examine each of those points separately.

The constitutional symptoms.—In foot-and-mouth disease there is usually a very marked increase of temperature, reaching from 104° to 107°. At Neosho Falls the temperature, as a rule, did not exceed what might reasonably be expected in health. Some of the perfectly healthy yearlings had a temperature of 103°, while that of most of the sick ones was below this point. One of the steers in the early stages of disease at Keith's showed 104.4° on March 9, which was about the highest point reached by any. In foot-and-mouth disease there is loss of appetite and difficulty of swallowing, but here the universal testimony was that the appetite had remained good throughout and there was no trouble in mastication or swallowing. In Illinois there were marked symptoms of digestive disturbance, and the disease was ushered in by diarrhea.

The mouth symptoms.—In foot-and-mouth disease there is an eruption of blisters on the mucous membranes of the lips, gums, tongue, and palate, which are numerous and painful. Often they unite with each other and form large patches, from which the covering becomes detached, leaving ulcerous patches of a bright red color and of great sensitiveness. It is almost impossible for animals in this condition to eat hay or other dry food, and it is necessary to support them with gruel. Such animals stand, making a peculiar and rather loud smacking noise with the lips and tongue, grinding the teeth and slavering profusely. In Kansas the mouth symptoms were much less severe than this, but two or three animals were reported to have had any salivation or any difficulty in eating hay. Some of the mouths presented erosions, which were mostly small, very superficial, and without any appearance of ulceration. I did not see a single blister, but a few of these were reported by those who visited the herds at an earlier date. The lesions which I saw in the mouths seemed to be due rather to a softening of the mucous membrane than to vesication; and I was assured that the appearances did not differ materially at the time of my visit from what they were when the veterinarians first saw them. In one or two animals there were large patches of thickened mucous membrane of a yellowish color, hard and difficult to detach. Healthy herds in the vicinity were visited, and in the mouths of these cattle were found discolorations and erosions very similar to, though less extensive than, those seen in the sick ones. In Missouri some of the cattle had the mouths involved to a greater degree than any I saw in Kansas, but others with equally bad feet had perfectly sound mouths. Here I saw pieces of mucous membrane becoming detached, but no blisters. Figures 1 and 2, Plate X, show the highly inflamed condition of the ulcers in the real foot-and-mouth disease.

The cattle in Illinois still had erosions in their mouths as late as April 24, which were identical in appearance with those I saw in Kansas. The steer which first came down with the disease on the Faunce farm, and which had consequently been affected about four months, showed these about as plainly as any animals I saw in Kansas. In foot-and-mouth disease the eruption disappears in from two to three weeks, and the animal is convalescent. Before proceeding to Kansas and Illinois the second time, I visited the herds at Portland, Me., which had been affected with foot-and-mouth disease. The contrast was very striking. Although the cattle in Maine had not showed the disease until the second week in February, they were on the 16th of April in apparently good health. There were no longer any sores in the mouths or on the feet. A week later than this I found cattle in Illinois that sickened in December and still had as marked mouth symptoms as could be found in any of the Western herds.

The feet symptoms.—The interdigital spaces and the coronet are the seat of the eruption in foot-and-mouth disease. Not only is there redness, heat, and swelling in these parts, but there is formation of blisters, loss of epithelium, and a secretion from the whole affected surface of the skin. The appearance of the feet with sheep and cattle having this disease is shown in Figs. 1, 2, and 3, Plate IX. Sometimes abscesses form beneath the horn, from which the pus may burrow and cause the loss of the hoofs, or even affect the ligaments and joints. But severe complications in the region of the foot do not occur except from this cause. With the cattle which I visited, the feet presented a very different appearance. Some of the limbs were separating, as a consequence of dry gangrene, half way between the fetlock and hock joints, with the skin of the foot still in perfect condition, though dead. In others the separation occurred at the fetlock, and in many others at the joints below, but not as a consequence of the burrowing of pus. Indeed, very little pus was to be seen in any of the feet. It is not rare to see the horn of one or both toes lost in foot-and-mouth disease, but it would be remarkable for the whole toe, including the bone, to slough off, as occurred so frequently here. I did not see a case where the hoof was lost without a loss of the bone at the same time. The complete death of the foot to the fetlock, or even higher, as occurred in all the worst cases in the West, is altogether unheard of in foot-and-mouth disease. While there was redness, heat, and swelling above the line of separation, I saw no appearance of blisters between the toes or around the coronet. A large proportion of the affected animals were simply lame, and had neither blisters nor sores about the feet. Finally, the disease was generally confined to the hind feet, or, if it attacked a forefoot, it was only after both hind ones were affected. Foot-and-mouth disease has no such decided preference for the posterior extremities.

The eruption on the udder.—In only one case that I have heard of in the West was there any appearance of an eruption on the udder of the affected cow. This was a cow belonging to Mr. Keith, the young calf of which died, as was supposed from the effects of the disease contracted from its mother. I am unable to account for the sores which evidently existed on the udder of this cow, not having seen her until they were nearly healed. There is also considerable doubt as to the cause of the calf's death. Certain it is that an eruption of blisters on the udder is an extremely common occurrence in foot-and-mouth disease (Fig. 3, Plate X). In the West, however, a considerable number of cows were affected, and but one had any symptoms of this kind.

Reviewing these symptoms, we can see that the disease which I investigated had few if any characters in common with foot-and-mouth disease. Among the whole number there was not a single animal which presented the typical characters of this plague. There did not appear to be a single animal which presented even the typical mouth symptoms, or the typical feet symptoms of that disease. The history, the characters, the symptoms, everything connected with the disease, led us to conclude, therefore, that it could not be the contagious foot-and-mouth disease.

CHARACTERS WHICH DISTINGUISH THIS DISEASE FROM FOUL-IN-THE-FOOT.

The disease known as foul-in-the-foot, and often called foot-rot, has its origin in the skin of the interdigital space. It begins as a superficial inflammation, which is followed by sloughing, ulceration, suppuration, the burrowing of pus, and the formation of sinuses. By this process the disease may gradually extend beneath the horn of the toes and toward the deeper parts of the foot, until the tendons, bones, ligaments, and articulations are involved. In extreme cases it may even extend to or above the fetlock joint. Steel, in his new work on the "Diseases of the Ox," sums up this characteristic of the disease as follows:

> Thus the pathological conditions of this disease are, at first, the existence of inflammation in the interdigital substance, which may be partially removed by sloughing, then the presence of pus beneath the hoof-horn, boring and forming simple sinuses, which extend outwards and burst on the surface. The patient is very lame, and the digits are separated from one another in a remarkable manner.

That is, foul begins between the toes, forms sores there, and these slowly extend by ulceration and the burrowing of pus. Neither in Kansas, Missouri, nor Illinois were any such pathological characters as these seen. There was sudden and complete death of a toe or of a foot, or in some cases of a leg as high as the hock joint; the disease showed no tendency to extend, but was limited by a groove around the limb, which soon became a crack, and the affected portion was sloughed off. There was no burrowing of pus, no ulceration, and when the lifeless portion of the limb had separated, the stump healed as readily as could be expected. The disease was dry gangrene beyond question, and dry gangrene is not produced by foul-in-the-foot.

Again, those who so confidently pronounced the disease to be foul, overlooked the gangrene of the tails, which was present in a large number of cases, and was most marked in those animals in which the feet were most severely affected.

Finally, the digestive disturbance and the lesions in the mouths were too evidently connected with the disease in the feet to be left entirely out of consideration.

It is surprising that two diseases having such different symptoms could be confounded, and the mistake of such a number of competent veterinarians can only be explained on the supposition that the examination was hurried and superficial, and that ergotism among animals has received but little attention in English-speaking countries.

OBJECTIONS WHICH HAVE BEEN URGED AGAINST THE THEORY OF ERGOTISM IN KANSAS.

When we first diagnosed the disease at Neosho Falls to be ergotism, we were met by the objection that ergotism could not occur without ergot in the food, and that this condition did not exist on the affected farms. It required but a few minutes inspection of the hay-racks, however, to satisfy the most skeptical that the hay at Keith's, Beard's, and Pribbernow's contained a large quantity of ergot in the wild rye which made up a considerable proportion of the forage. And subsequent examination has proved its existence nearly everywhere that this disease occurred.

The second objection was that ergot did not produce dry gangrene in animals; and this statement has been repeated again and again by professional men who certainly ought to have known better. The quotations from standard authorities as to the effects of ergot and the historical compilation contained in other sections of this report will be sufficient, I believe, to satisfactorily dispose of these assertions.

Again, it was said that it required enormous quantities of ergot to produce appreciable effects on cattle, and even if it could in that case cause dry gangrene, the quantity found in the Kansas hay was totally insufficient to account for these results. To this I reply that ergot in different seasons is known to differ widely in its poisonous qualities; that certain conditions, such as extremely cold weather and deficiency of drinking water, undoubtedly increase its effects in a very important degree, and that, finally, we do not know how much is actually necessary to cause dry gangrene. Careful estimates of the quantity of ergot in the hay in Illinois, Missouri, and Kansas show that these cattle must have taken with their food from 3 to 4 ounces of this poison each day. The dose of ergot recommended by standard veterinary authorities as safe for medical purposes is about one ounce for grown animals, but it is not expected that this would be used for more than one or two days together. The diseased cattle, therefore, had taken from three to four full doses of ergot a day, and continued this for days and weeks. Considering that the action of ergot is to diminish the caliber of the blood-vessels, that the gangrene of the extremities is directly traceable to deficient blood supply, and that atmospheric cold also has a marked tendency in this direction; that, in addition to all this, the drinking places were frozen over, and the holes that were cut through the ice were only kept open a short time each day, it would appear that the conditions were very favorable for the development of ergot poisoning.

Then it was reported that the outbreak in Osborne County was certainly the same disease, and that the cattle had been pasturing on green rye and consequently could not get ergot. A few inquiries brought out the fact, however, that the rye pasture had only been in use for three weeks, while the disease had appeared at least six weeks previously. There was no reliable information as to what these cattle had been eating before the appearance of the disease, and the forage was not examined by any competent person.

"If this disease is due to ergot poison," says one gentleman, "why then is this the first outbreak, since the Kansas farmers have fed this same kind of hay to their cattle from the first settlement of the State?" But who knows that they have ever before fed hay containing as much ergot? In Europe the enzootics of ergotism have at times been a century apart, and it is a well known fact that it is only in occasional years that these enormous quantities of ergot are produced. Then how can it be known that this is the first outbreak of the disease in Kansas? Cattle have frequently suffered with the same symptoms in New York, Pennsylvania, Iowa, and Missouri, and why not also in Kansas? Who outside of the immediate vicinity of the suffering herds would have heard of the outbreak under consideration had it not been for the mistaken diagnosis that led the country to fear the presence of a dangerous contagious disease?

Again, would not this reasoning apply to any supposed cause of the disease as well as to ergot? No matter what produced the disease, if such an outbreak has never occurred before it might be said with just as much force, "Why, then, is this the first outbreak?"

"Again," the same gentleman goes on to say, "on Mr. Goodrich's farm, where the disease prevails, the lands are improved by cultivation, and there is no ergotized rye in his hay. Yet out of 96 cattle, 40 head of young stock are reported affected with the disease." This fact was the most troublesome of all I had to contend with in making my diagnosis, and I appreciated its importance perhaps as much as my critics could have appreciated it. The gentleman's statement is not absolutely correct, however, as there was a small quantity of ergoted rye in the hay; but still there was so much less than was seen at the other farms as to make it impossible to explain why the cattle here should be affected even to a greater degree than elsewhere. In my preliminary report I explained this by saying that wild rye was known to grow in patches, and that, consequently, hay that was being fed at one time could not be considered as exactly the same as that fed three months before. The apparent discrepancy in this case has since been explained, however, in a much more satisfactory manner. Some time last fall Mr. Goodrich bought two stacks of hay of Mr. Keith, and it was this hay that he had been feeding to his cattle up to the time of the outbreak of the disease. When this fact was learned the whole matter became perfectly clear, and what at first appeared the greatest objection to the ergot theory turned out to be one of its strongest supports.

Then Mr. Beard is mentioned as having fed 75 head of cattle all winter on hay full of ergot, and escaped with but 5 diseased animals. Mr. Beard, however, had fed his cattle twice a day on corn-fodder, that is, on corn which had been shocked but not husked, and as a natural consequence his cattle ate very much less of the hay.

"Stranger still for the ergot theory, Mr. Pribbernow fed 195 cattle on millet hay and corn-fodder, and he has 14 of his young stock affected." Here, again, the zeal of the gentleman to make out a case against the ergot theory has led him to make statements which are not correct. Mr. Pribbernow had some very badly ergoted hay, which he showed to me, and told me that he had been feeding it to his cattle; and, indeed, there was plenty of evidence that this was the case from the condition of the feeding yards and racks. It is a fact, however, that 54 yearlings were fed on millet hay, oats, and corn-fodder in addition to the hay, and that not one of these was affected. The older cattle had been fed more exclusively on the hay, and it was among them alone that the effect of the ergot was seen. These facts I noted down as they were related to me on the spot by Mr. Pribbernow.

"Another puzzle is presented by Mr. Keith buying 63 head of young stock from Mr. Davis on the 15th of December, and on the 23d nearly all were down with the disease. Keith's hay contains ergotized rye. Davis has had no sickness in his herd." This statement is also very incorrect, and yet it contains a reference to the one unsolved difficulty connected with the Kansas outbreaks. The 63 head of cattle were purchased December 10, and as the first cases of sickness on this farm did not occur until the 23d or 24th, and as at the 1st of January there were still less than thirty cases all told on the farm, it is plain that these animals had sufficient time to contract the disease after their purchase.

The difficulty in regard to the ergot theory at Keith's was in connection with another lot of cattle bought about the 15th or 20th of December. This lot consisted of 6 yearlings and 2 cows, some of which Mr. Keith asserts were sick within three days and all within eight days, and that they were not fed upon hay during that time, but upon mowed oats and corn-fodder. He admitted, however, that there was probably hay in the racks to which they had access. There was much doubt as to the days on which these cattle were first seen to be lame, and as to how severely they were affected. It is also impossible to say, at this time, on what they had been fed previous to their purchase. This difficulty, however, does not compare with that felt at first in regard to the Goodrich herd, and as the latter was satisfactorily explained at the last minute, it is not at all improbable that there are some unknown facts in regard to the 8 cattle in question that would explain this case just as satisfactorily.

I have reviewed above the chief objections that have been advanced to show that the disease in Kansas could not be ergotism. It is unnecessary to add that they are mostly of the nature of captious criticism. The malady had been pronounced foot-and-mouth disease by somè and foot-rot or foul by others, and these gentlemen found it desirable to make out at least an apparent case against ergotism. In other sections of this report I have given abundant evidence to show that it could be nothing but ergotism, and the plates herewith presented, which were carefully prepared by a competent artist, are sufficient to prove this beyond doubt to any one who understands the pathology of these different diseases.

THE NATURE, CHEMICAL COMPOSITION, AND ACTION OF ERGOT.

The substance known as ergot is one of the stages in the life history of a fungus which has been named *Claviceps purpurea.* The term ergot was applied to it by the French from its fancied resemblance to the spur of a cock. The place which this fungus occupies in the plan of nature may be understood from the following table, which is taken principally from the classification proposed by Sachs:

Vegetable Kingdom.	Group.	Class.		Order.	Family.	Genus.
		Protophyta.			Gymnoascus.	
		Zygosporeæ.			Discomycetes.	Claviceps.
	Thallophytes.	Oosporeæ			Ersipheæ.	Cordyceps.
	Muscineæ.	Carposporeæ.	Containing Chlorophyll.	Ascomycetes.	Tuberaceæ.	Byssothecium.
			Without Chlorophyll	Ascidiomycetes.	Pyrenomycetes.	Fumago.
	Vascular Cryptogama.			Basidiomycetes	Lichens.	Pleospora.
	Phanerogama.					Sphaerella.
						Laboulbenia.

The growth of the claviceps begins by the germination of conidia or spores of this fungus, which have been carried by currents of air or other means to the flowers of the grasses favorable for its development. These conidia or spores, as the case may be, germinate in contact with the external surface of the ovary while this is still in an early stage of its growth, and form a mycelium which penetrates the walls of the ovary, and, as a rule, respects only the summit. It thus by degrees substitutes itself in place of the tissue of the ovary, and, consequently, preserves to a considerable extent the form of this organ. It bears on its summit the stigma, while its external coat is traversed by deep grooves and irregular cavities. (In Plate VIII, Fig. 1, is seen the normal ovary of the rye plant; Fig. 2 shows the same invaded by the claviceps.)

In this stage of its existence it has been called the sphacelia. As it develops it takes entire possession of the ovary, obliterating its cavity, and preventing the development of the ovule. The ovule may be either entirely absent or it may be present in an imperfect form, but does not develop into a seed. The mycelium produces at the surface of the ovary a large number of oval corpuscles, which are called conidia, and which falling upon other flowers may germinate and again produce the mycelium or sphacelia form of the fungus. (Pl. VIII, Fig. 3, which is a cross-section of the ovary, now called sphacelia, shows these conidia at its borders.)

The sphacelia, however, is not the ergot form of the fungus. At the base of this is produced a hard substance with a black or dark violet surface, and white or grayish within, which is the true ergot or sclerotium stage of the claviceps fungus. In the earliest period of the development of

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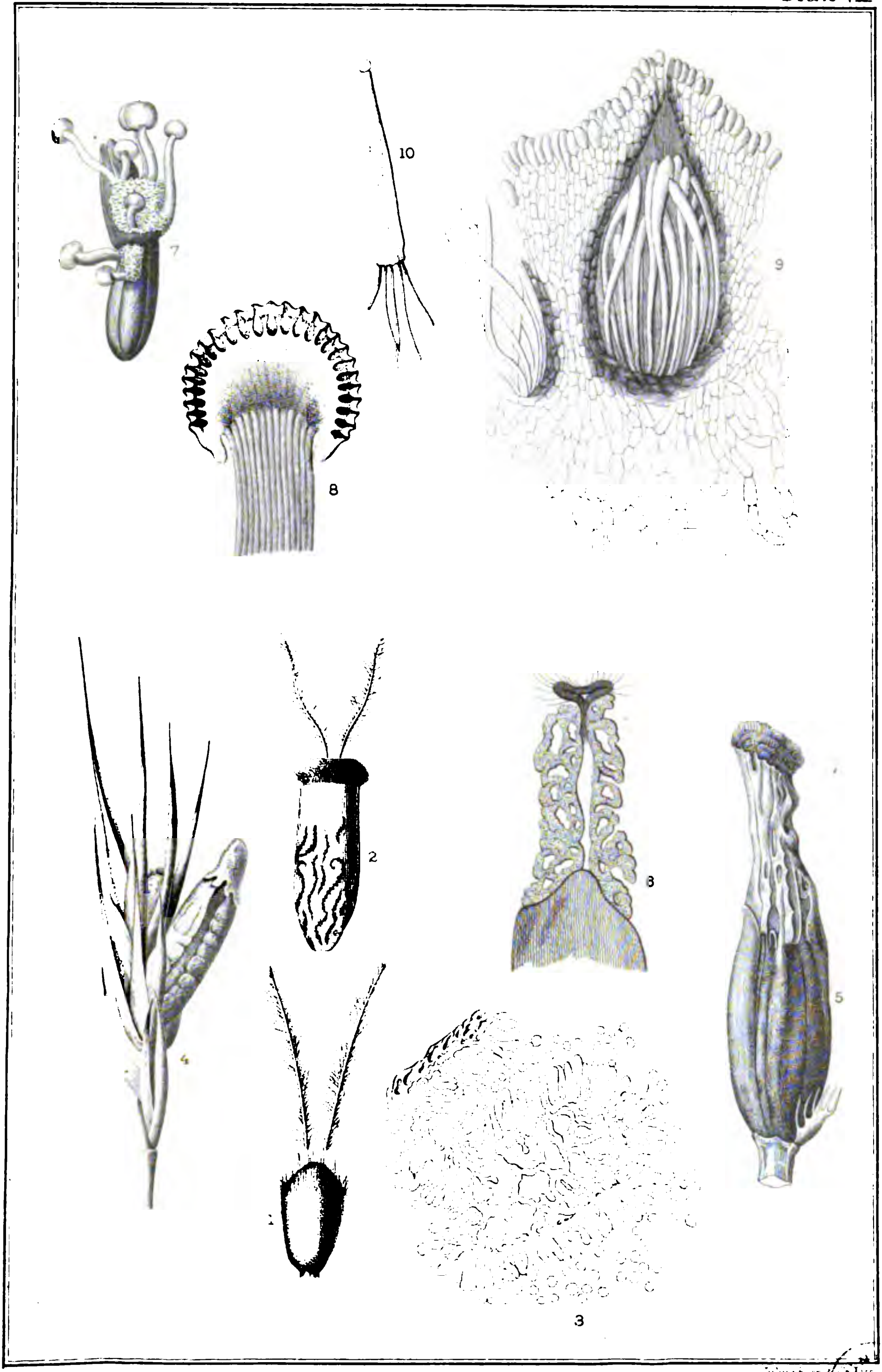
Plate VIII



DEVELOPMENT OF ERGOT

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the sclerotium this is entirely covered by the sphacelia, but it gradually increases in size and pushes the sphacelia before it until the latter is raised entirely beyond the floral glumes, and is supported on its summit (Pl. VIII, Figs. 4, 5, 6). The sclerotium, or ergot, continues to increase in size and length, and the deformed ovary or sphacelia adheres to it for a long time, and may even be found in a considerable proportion of the specimens as seen in hay or mature grain after curing. In these the form of the stigma can be readily made out in most cases by the use of a low power lens.

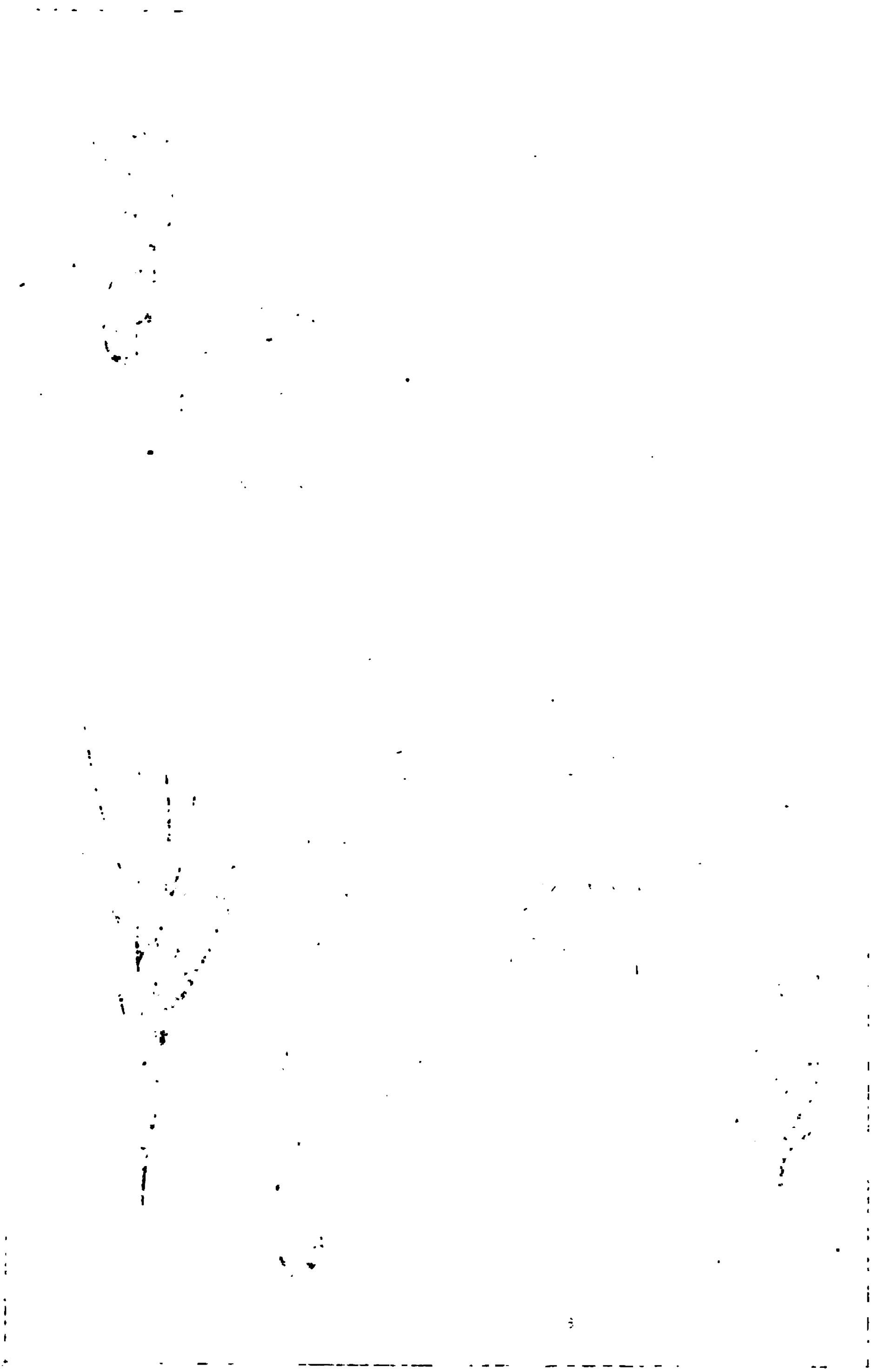
Ergot is not therefore a diseased seed; on the contrary, it develops entirely below the ovary and prevents the formation of the seed. It is entirely a fungus growth, and is the resting stage of the organism. It contains little or no starch, and its microscopical structure is that of the sclerotic mycelia. The sclerotium is looked upon as a hard compact mass of imperfectly developed mycelia. It appears to be about a month from the time the fungus invades the ovary until the ergot is fully formed.

The ergot is the dormant form of the fungus, and remains in this condition until autumn, or usually until the next spring. If at this time it is in contact with the damp ground it germinates and stromata grow from its surface (Pl. VIII, Fig. 7). These consist of a long stalk and a globular head, and become perfect fruiting fungi. In the head a large number of flask-shaped perithecia are formed (see Pl. VIII, Fig. 8), which are filled from the bottom with a number of asci (Pl. VIII, Fig. 9), each of which contains several slender filiform spores (Pl. VIII, Fig. 10).

When the spores reach the young flowers of rye, red top or other nearly allied grasses, they germinate and form a mycelium which invades the wall of the ovary and again produces a sphacelia. With this the cycle of development of the fungus is completed and we probably have its entire life history. The meteorological conditions most favorable for the production of ergot are not well known. It has been asserted that it only appears in large quantities in rainy seasons, but others believe that moisture has little or no influence on its development. It is also uncertain whether more than one species of claviceps is concerned in the production of ergot in the different varieties of grasses. The ergot of the red-top hay in Missouri and Illinois produced identical effects with that in the wild rye of Kansas, and it would therefore appear that the physiological effects are substantially the same even though the species growing upon these two plants may be different.

The grains of ergot of rye are from half an inch to over an inch in length, and from one-fifteenth to one-sixth of an inch in diameter; they are nearly cylindrical, sometimes slightly ribbed and furrowed, and often have irregular fissures; they are curved, and taper toward the ends. The color of the surface varies from dark violet to blue-black or black; the interior is white, often tinted with violet. The ergot of wild rye, blue grass, and red top has the same general appearance, but the grains are smaller. In red top many of the grains are so small that they are only recognized with difficulty by the unaided eye. Sometimes the taste is pronounced and disagreeable; but the ergot in the wild rye of Kansas, where the outbreaks of disease occurred, was almost or entirely without taste, and certainly was in no sense disagreeable when masticated.

Chemical composition.—Ergot is a very complex material when considered chemically, and although it has been studied by many competent chemists, there is yet much doubt as to the nature of a number of



DEVELOPMENT OF [illegible]

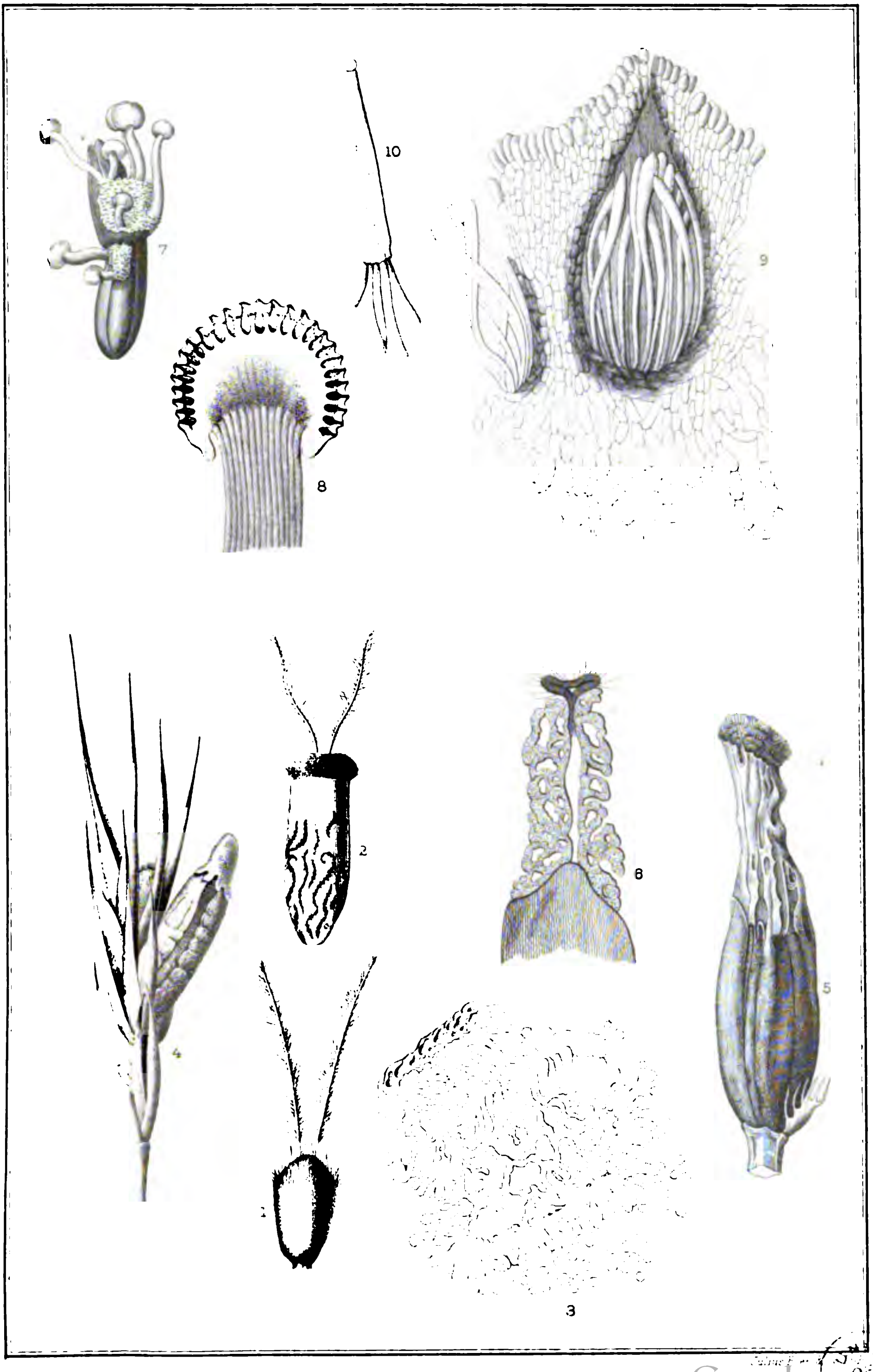
sphacelia, but it … the latter is … supported on its … In these the … by the use of

… It is … ergot is …

… If at this time it … and stromata grow … a long stalk and a … (see Pl. VIII, Fig. 8), … of asci (Pl. VIII, Fig. 9), … spores (Pl. VIII, Fig. 10). … rye … or other … sphacelia, which … with … the cycle … completed and we … logical conditions … production of ergot are not well known. It has … appears in large quantities in rainy seasons, … little or … concerning its devel- … certain whether more than one species of … varieties of … and it would … substantially the … same even … may be different.

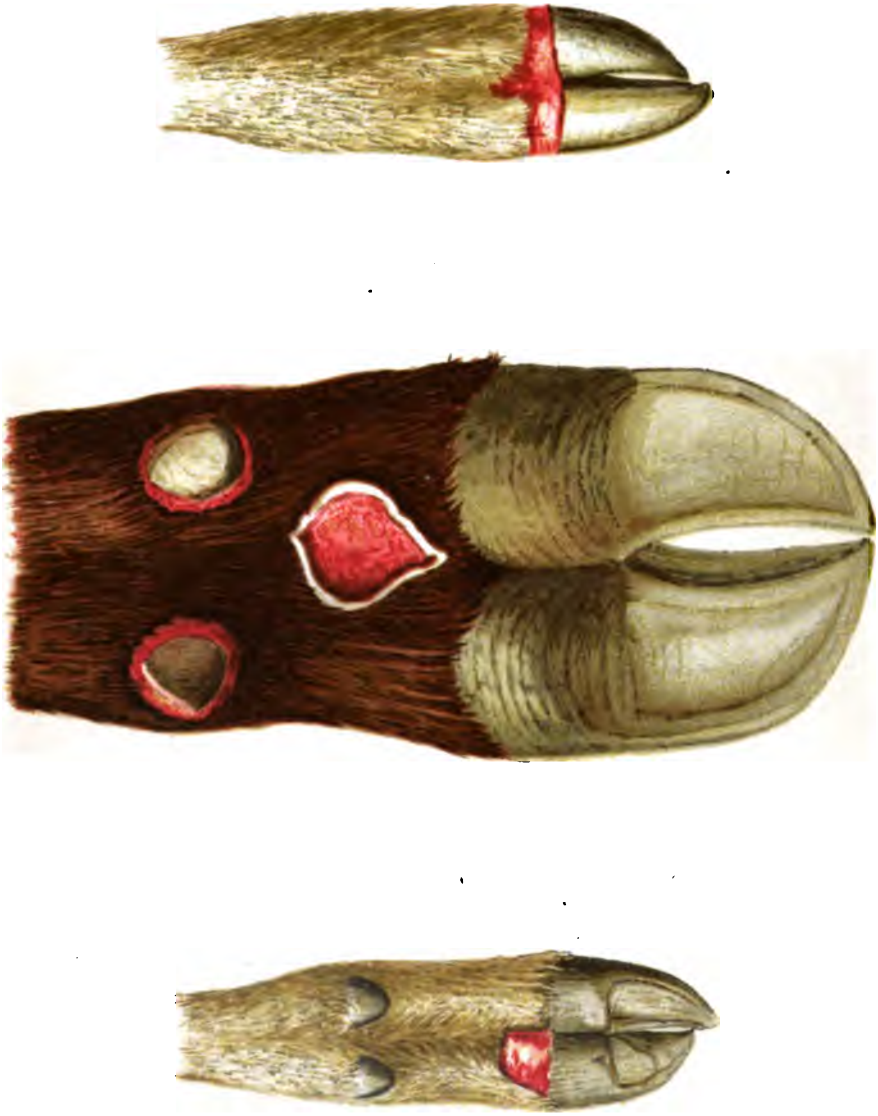
The grains of … to over an inch in length and from … they are nearly cylindrical … narrowed and often … toward the ends. … irregular … black or brick; … of the surface … The ergot of wild rye, … is white, … appearance, but the … and red … has … so small that … with … naked eye. … red … organized with … that the ergot in the … times … is … wild rye … Kansas, where the … case occurred, was almost … without taste, and … no sense disagreea- … ble when …

Chemical composition.—Ergot is … material when con- sidered … and although it … studied by many compe- … chemists, there is yet much con- … nature of a number of



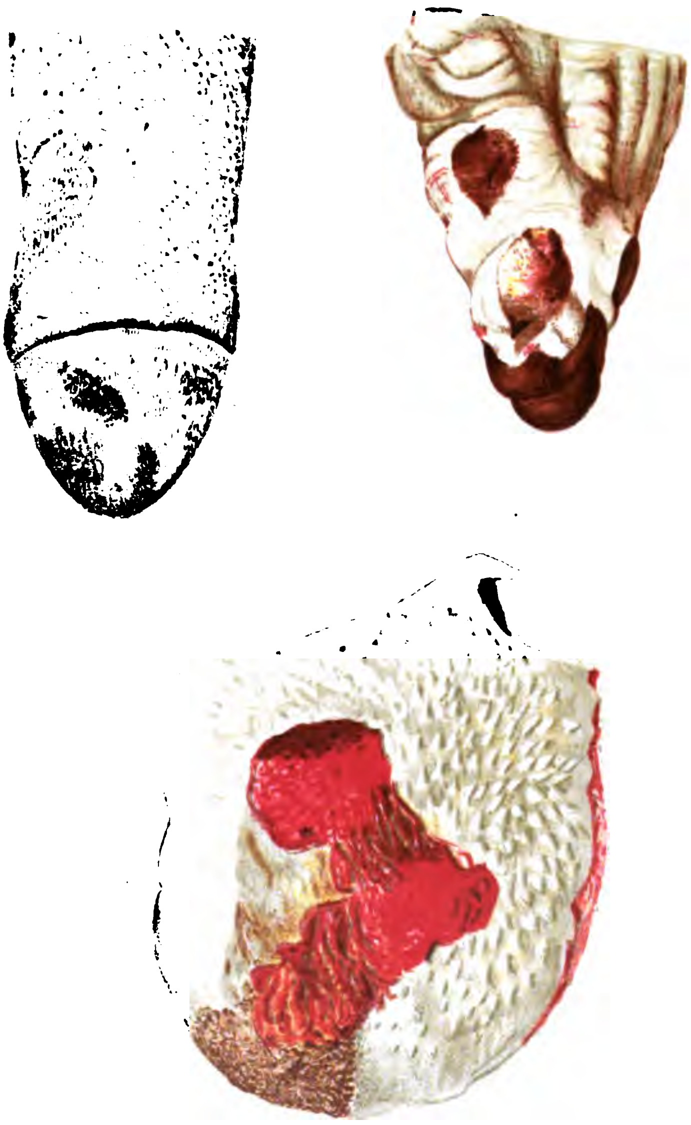
DEVELOPMENT OF ERGOT

Plate IX



FOOT & MOUTH DISEASE [illegible]

Plate X



Julius Bien & Co. Lith.

FOOT & MOUTH DISEASE (WALLEY)

UNIV. OF MICH.

the substances which have been found in it. About 35 per cent. of its weight consists of a thick, fluid, fixed oil, which is now believed to be without medical properties. Two non-crystallizable alkaloids have been described and called, respectively, *ecbolina* and *ergotina*; and one crystallizable which has been designated as *ergotinine*.

Sclerotic, *ergotic*, and *fuscosclerotinic* acids have been isolated. There is also a question of a peculiar ammoniacal base variously stated to be *methylamine*, *trimethylamine*, and *propylamia*. A mucilaginous substance called *scleromucin* and several other products of doubtful nature have been recognized. It has not yet been demonstrated which of these bodies constitutes the active principle of the drug, or whether the physiological effect may not be due to a number acting together.

The one point on which most of those who have studied ergot have agreed is that water extracts the medical properties, and this seems to be about the extent of our reliable information in regard to this department of the subject.

The action of ergot on the animal body.—The action of this poison in large doses is very clearly given in the following extract from Dr. H. C. Wood's Treatise on Therapeutics, Materia Medica and Toxicology:

> According to Diez [quoted by Stille], the principal effects of poisonous doses of ergot are in the lower animals profuse salivation, vomiting, dilatation of the pupils, hurried breathing, frequent pulse, cries, trembling, staggering, paraplegia, sometimes diarrhea, sometimes constipation, prostration, urgent thirst, convulsions,* and death. Mr. Samuel A. Wright, in a series of experiments (*Edinburgh Med. and Surg. Journ.*, Oct., 1839, vol. lii), noted, when the medicine was given by the mouth, symptoms similar to those just spoken of; the paralysis was much more marked than the spasms. Late in the poisoning, the heart's action became irregular and intermittent, and the pulsations, which had been rapid, grew slow and feeble. In some cases the special senses seemed to be destroyed, and coldness of the surface was a very prominent symptom. Mr. Wright also injected a strong infusion of the drug directly into the torrent of the circulation. Death was in some cases produced in nine minutes, the symptoms being immediate dilatation of the pupils, great increase in the rate of the cardiac pulsations, paralysis, and convulsions. When the fatal result was not brought about in so short a space, great anæsthesia of the surface was noted a considerable time before death; coldness of the surface and paralysis of the special senses were also present in some cases. In Dr. Kersch's experiments (*Betz's Memor.*, vol. xviii), the concentrated infusion was injected into the jugular vein; the coldness of the surface was especially noted, and also great muscular rigidity. Upon rabits, according to the researches of Wright, ergot acts very feebly. In birds, as represented by chickens, turkeys, and pigeons, it causes symptoms analogous to those produced in mammals, as is testified by Tessier and by Gross, both quoted by Stille, and by Bonjean (*Traité de l'Ergot de Seigle*, Paris, 1845.)
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> The above summary of the general symptoms caused by poisonous doses of ergot shows that the phenomena are mainly paralytic in their nature; but, although an enormous amount has been written about the drug, we have very little knowledge as to the immediate causes of the paralysis. Since both Wright (*loc. cit.*, pp. 320, 321) and Köhler have found that the voluntary muscles are not affected by ergot, it would seem that the nervous system must bear the brunt of the poison. Eugene Haudelin is said to have shown that the peripheral nerves are not affected, and the experiments of Köhler have confirmed this so far as concerns the motor nerves and the watery extract of ergot. He found, however, that those portions of the drug not soluble in water appeared to increase the excitability of the peripheral efferent nerves, and that upon the peripheral sensory nerves both portions of the ergot acted as a feeble depressant. On the whole, it is probable that the chief action of the drug is upon the nerve centers.

The following experiments of Tessier also indicate the active nature of the ergot poison (*Memoire sur les effets du seigle ergoté. Hist. Soc. Roy. de Med.*, 1777, 1778, Paris 1780, vol. ii, pp. 587–615):

These experiments were instituted with hygienic precautions upon a number of animals. Of two ducks fed upon ergot, one, the female, died in nine or ten days. It had consumed one ounce and three drachms of ergot. There was a large violet spot on the beak, the covering epidermis was raised up by a collection of dark, fetid blood. The male died in fourteen days with the beak similarly affected; there was also drooping of one wing which showed two regions of inflammation, one in the fold and the other on the first phalanx. It had consumed 2 ounces and 6 drachms of ergot. A turkey was fed 8 ounces 4½ drachms of ergot within twenty-two days. The autopsy revealed inflammation about the beak, but none of the feet and wings. A pig six weeks old died at the end of twenty-three days after receiving 1 pound and 12 ounces of ergot. The autopsy revealed swelling of the four feet especially at articulations, which were a reddish violet color. The ears were livid, there was gangrene of one side of the head and various internal inflammatory lesions. The articulations of the feet with the legs being uncovered there was seen, particularly with the posterior limbs, a thick, black, and fetid liquid. The animal previous to death had been able to support itself better on its fore than on its hind limbs. A six-months-old pig died after being fed during sixty-nine days upon a total of 22 pounds and 6 ounces of ergot. The autopsy revealed various internal inflammatory lesions, several violet spots on front and hind legs, the end of the tail dark violet, and ears livid. The two first phalanges of the right anterior foot were gangrenous and dry, especially near the articulations. The bones themselves were tinted brown. The same parts of the left foot were gangrenous but not so far advanced, as the bones were not altered. Upon each calcaneum there was a livid spot, larger on one than on the other. During life there was on the twentieth day a purulent discharge from two cavities in the articulation of the right foot; these were soon covered with a crust. The limb remained cold. On the forty-second day the corresponding joint of the left anterior leg developed a tumor which by the fifty-eighth day became an open sore. Both legs were cold and swollen, dry, insensible, and portions of the muscles became detached. The animal was no longer able to walk.

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* Pereira states that convulsions were not present in the experiments of Diez.

Salerne, cited by Read, gave to a small male pig barley mixed with half its weight of ergot. At the end of fifteen days the legs became red, secreted a yellowish and fetid humor, the skin of the back and beneath the abdomen became black in color. This food was continued for fifteen days and then replaced by some free from ergot. The animal died four days later; there was no gangrene of the feet. Read fed a pig three months old for fifteen days with ergoted wheat mixed with bran. Gangrene seized the left ear on the seventeenth day and it dropped off. The pig died two days later with convulsions. A gangrenous spot was found on the liver. (A. Tardy. *De l'Ergotism*, Paris, 1858.)

Fleming, in his Manual of Veterinary Sanitary Science and Police, (Vol. I, p. 65), says: "The ergot on rye, wheat, &c., has also given rise to extensive disease in man and animals, including birds, marked by convulsions, paralysis, dry gangrene of the limbs, loss of hair and horn, and other strange phenomena."

M. Tabourin, in his *Nouveau Traité de Matière Médicale de Thérapeutique et de Pharmacie Vétérinaires*, Paris, 1866, gives the following description of the action of ergot (pp. 448 to 450):

> The effects of ergot of rye should be divided into medicinal and toxic.
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> *Medicinal effects.*—The action that ergot of rye exercises on the natural surfaces and on the denuded tissues has been very little studied with animals, but appears to be slightly irritating; with man it has been noticed that the aqueous extract arrests capillary hemorrhages with considerable rapidity, and that it has a manifestly astringent action on denuded tissues. In the digestive tube the effects are but little marked when the medicine is given in small doses; it is only when the quantities in-

gested are considerable that vomiting occurs with carnivora and a serious irritation of the intestines with all animals. In regard to the dynamic or general effects produced by the ergot of rye in medicinal doses, when its active principles have been absorbed, they are almost unnoticeable with healthy animals and have been only very imperfectly studied up to this time. It follows, however, from the trials undertaken by various authors on the greater part of the domestic animals, that this medicine produces with them as with man two effects somewhat opposed to each other: a very pronounced sedative action on the circulatory center, and an energetic stimulation of the nervous centers and particularly of the posterior portion of the spinal cord. We will return to these two culminating effects of ergot of rye in connection with the toxic action that it has on the organism which we are now about to study.

Toxic effects.—The poisoning of animals by ergot of rye is called ergotism. It may occur at the end of a longer or shorter time, according to various circumstances and particularly according as the ergot is given alone or mixed with the food. In the former case, it occurs after a few days with birds, and after weeks or even months with mammals, according to the size of the doses and the time between them. In the second case it is much slower still, and when its existence is manifested by apparent phenomena the destruction of the organism is already consummated and there is no means of providing a remedy for it. This is a remarkable example of chronic or slow poisoning.

The characteristic signs of ergotism are of two varieties. One of these is due to the narcotico-acrid and exciting action that the ergot exercises on the nerve centers; the other is due to the sedative action that it produces on the heart. When the former predominates, as has been observed with certain epidemics with the human species, the ergotism is called convulsive; when, on the contrary, the second is more pronounced the ergotism is called gangrenous. It is difficult to establish this distinction with animals where the signs of the two varieties are mixed in nearly equal proportion as we shall demonstrate.

1. *Solipeds.*—Of all the domestic animals, the solipeds are the least exposed to poisoning by ergot of rye, because oats, the grain they receive most often, is rarely affected with this alteration. Only two authors, MM. Hertwig and Parola, have made experiments on solipeds with ergot of rye. The former administered three and one-half kilograms (7.7 pounds) of this substance to a horse in the space of 24 days; he observed some nervous phenomena and a great depression of the circulation, but no appearance of gangrene. The latter gave ergot of rye to a mule, affected with chronic coryza, for six days in the dose of one to two ounces a day. There was slowing of the circulation, decreased temperature of the body, difficulty of respiration, loss of appetite and strength, general depression, muscular trembling, slight swelling of the knees toward the end, &c. The subject was destroyed. The dischage from the nose had disappeared.

2. *Large ruminants.*—Poisoning of large ruminants by ergot is more common than that of solipeds because these animals receive quite often, as a supplementary ration, the rejected grains coming from the thrasher or from screening, which always contain more or less ergot of rye and of other grains. With the large ruminants the convulsive phenomena are not seen or are not very apparent; but the depressive effects on the circulatory system are, on the contrary, very marked. Besides, the health is maintained without serious disturbance during weeks and even months if the ergot is taken with the food. Only the extremities lose little by little their natural warmth, as is noticed with the ears, the tail, the lower part of the limbs, &c. The digital region, and sometimes even the metacarpal and metatarsal regions, as M. Decoste has observed, are smitten with dry gangrene. In this case, the parts lose their warmth, their sensibility, become hardened and mummified, and soon separate without pain from the parts which have still remained living.

3. *Small ruminants.*—It is known that the sheep may, like other animals, feel the noxious influence of ergot; but science is wanting in precise documents concerning this ruminant and the goat.

4. *Pigs.*—It follows from some experiments made on these animals by Tessier, that ergot of rye poisons them after a greater or less time according to their force of resistance. There is seen in the first place vertigo, unsteadiness in standing, a tottering walk, moaning, swelling of the eyes, &c.; then the ears, the tail, the lower part of the limbs, lose their warmth and vitality; soon appear livid spots, which afterward become black and gangrenous, and are the beginning point for the separation of the mortified from the living parts.

5. *Dogs.*—M. Dieu has given ergot of rye to dogs in the dose of 15 grams (half an ounce) a day. The animals were soon taken with nausea, bloody diarrhea, a nasal discharge also colored with blood, depression, weakness, and soon drop into a frightful marasmus. The experiments not having been pressed to the end, the phenomena of dry gangrene could not be observed.

6. *Fowls.*—These little animals are most exposed to the poisoning under consideration, because they often receive for nourishment the residue from cleaning grain

which always contains more or less ergot. The first signs of this poisoning are loss of liveliness, indifference to surroundings, and great dullness; then there is vertigo, drooping of the wings, &c.; finally appear more characteristic signs—a bloody discharge from the nostrils is seen; the crest becomes black, shrunken, and mummified; the beak dries and is detached; the same course is soon followed by the tongue; the feathers lose their luster and fall out. Death results soon after these symptoms are seen.

To recapitulate, the most ordinary signs of ergotism with the various animals are as follows: Dulness, fixed expression, vertigo, dilated pupils, intoxication, coma; in the beginning muscular tremblings, then convulsive shocks, tetanic attacks, particularly in the posterior members which afterward become weak and paralyzed, unsteady position while standing, slow and difficult walk, &c.; general weakness, progressive emaciation; pulse slow and weak, skin cold; hair dull, limbs, ears, horns, and tail lose their natural warmth; sero-mucous and sometimes bloody discharge from the nostrils, cold swelling of the limbs; black spots, livid patches, gangrenous sores; dry gangrene of the crest, of the beak and of the tongue of birds, and of the ears, the tail, the phalanges. the limbs, which become detached little by little and piece by piece from the trunk without inflammation or pain, &c.

Lesions.—The digestive tube is more or less intensely irritated, the viscera are flabby and softened, the muscles semi-gelatinous, the blood fluid, violet colored, the interior of the vessels red as in putrid diseases &c.

M. Verheyen, in his article on ergotism in the *Nouveau Dictionnaire pratique de Médicine, de Chirurgie et d'Hygiène Vétérinaires*, gives the following account of the effects of this poison:

Symptomatology.—In spite of the numerous experimental studies of which the ergot of rye has been the object, its effects on the organism are far from being sufficiently understood to enable us to write in a complete and connected manner the part of the medical history of ergotism relating to the symptomatology and pathological anatomy. The mode of action of ergot on the economy is only presented so far in a fragmentary state; the acquired knowledge does not permit the tracing of a physiological chart of all the phenomena produced by this agent. A large number of experimenters, particularly among the modern ones, have only produced an acute intoxication, of rapid progress, which leaves in obscurity the evolution, the graduation, and the succession of the morbid phenomena; in a word the progress of natural ergotism resulting from the introduction into the economy of small but long continued doses of the toxic substance. History mentions destructive epidemics, and others which have been relatively mild; this difference can only be due to predisposition and to the abundance of ergot. Rye is rich in it in the calamitous years; the high price of cereals, and of all kinds of provisions, prevents the poorer classes from procuring sufficiently nutritious food. There, consequently, follows a constitutional debility and anemia, which singularly favors gangrenous and convulsive disorders. The observation, so precise, of M. Decoste, as regards the hygienic diet to which the cow, which was the subject of it, had been submitted, the conditions under which the epizootic of the State of New York appeared, proves that misery constitutes a predisposing cause not less energetic for animals than for man. The experimenters have not taken into account these constitutional modifications which increase the susceptibility to the poisonous agent, and give to ergotism a new symptomatic expression. Here, it appears to us, is found the key to the numerous contradictions that are noticed when the experiments are compared with each other. To cite only a single example concerning the bovine species, we see that Riemann did not succeed at the end of eight days in provoking the least abnormal phenomenon, and that Wahlin produced no other noticeable symptom than constipation. The authors who have observed cases of ergotism with animals during the course of an epidemic may be correct when they mention the fact very summarily, and limit themselves to a statement that the phenomena were absolutely similar to those presented by man. This identity justifies, to a certain extent, their laconism, and the epidemic conditions explain the differences obtained by experimenting during the epidemic periods and out of these periods. During the epidemics the rule relative to the gangrenous form in the south and the convulsive in the north is applicable to artificial ergotism. A final remark, perfectly justified, and which has been little if at all considered by the experimenters: Tessier, who brought a high order of intelligence to the elucidation of the history of ergotism, asserts that all animals show a very great repugnance to take ergot voluntarily; this is so insurmountable for some individuals that they will die of hunger rather than touch it. Consequently all were far from being assured that the ergot offered was really consumed.

Having made these observations, let us take up the symptomatology of ergotism—they will excuse the incomplete sketch that we trace.

These morbid phenomena are very inconstant during the period of invasion. Sometimes they indicate a lesion of the cerebro-spinal apparatus, at other times the digestive tube is invaded, at still other times the symptoms proceed from the circulatory system. This variability is common with fowls, with pigs, and with carnivora. The predominance of the cerebro-spinal affection manifests itself in various degrees of intensity; it may be arrested after development when the exciting cause, the alimentary use of ergot of rye, has ceased. In the first place, there is vertigo; the animals stumble as if they were intoxicated; they lose their equilibrium, fall on the side, and remain in a state of drowsiness, which is not dissipated even when they arise. The hair and feathers lose their luster; the temperature of the skin is lowered; there is anæsthesia, the insensibility following a condition of hyperæsthesia; this alternation affects also the sight and the hearing (Wright). It is unmistakable in the canine species; the pupils are constantly dilated. The symptoms of narcotism that we have just enumerated persist or are interrupted by convulsive phenomena, sometimes of the limbs only, and sometimes of the whole body. The general convulsions are characterized by tetanic epileptic attacks usually followed by temporary paralysis of the posterior parts. The suffering is sometimes so intense that it is manifested by plaintive cries and contortions. The nervous attack over, the animal falls again into a condition of apathy or drowsiness. If the spasm is limited to the limbs, there remains after the attack a contraction which persists for a certain length of time.

These phenomena which characterize spasmodic ergotism have an indefinite duration. Death may occur after a few hours or a few days as a result of a paroxysm, or the disease may be more prolonged and take a chronic form. The nutrition suffers; the animals become thin in spite of the appetite, which, however, is irregular, and finally a convulsion at last destroys them in an advanced condition of marasmus. The circulation is abnormal, the pulse is slow, accelerated but afterwards retarded; the arterial and cardiac contractions are spasmodic.

The participation of the digestive tube, which may be either the principal or the accessory cause of the cerebro-spinal affection, is announced by nausea, pharyngial spasms, vomiting, diarrhea, sometimes followed by an insatiable hunger. If this is satisfied the food does not alleviate the hunger, for it causes convulsions. In the south all these symptoms may be preceded by gangrenous accidents; the latter may also precede when the circulatory lesion is the first to occur. With the gallinaceans the crest becomes cold, takes a violet or black color, shrivels, and dries; these phenomena are also quite constant in the north, but the dessication of the beak, sometimes of the feet, constitutes an alteration exclusive to the south; gangrenous patches also cover the abdominal walls (Millet). In gangrenous ergotism of the palmipeds, besides the beak, there is sometimes seen mortification of the point of the tongue (Tessier), and of the interdigital membrane which is discolored and becomes dry and brittle; then the digits are lost (Decoste). With mammals the gangrene attacks the lower part of one or several limbs, the ears or the tail; these parts become red as if they were the seat of an erysipelatous inflammation; the color changes to violet, to blue, or to black; they become mummified and detached when the convulsive paroxysm has not destroyed life before the completion of the work of elimination. While this is occurring the loss of flesh progresses and marasmus comes on, then, finally, a convulsive movement which destroys the patient. The mummification also attacks isolated muscles and in very exceptional cases the dry form of gangrene is associated with the humid form (Tessier). The pulse remains small, feeble, slow, or indeed it becomes accelerated, febrile, and precipitates marasmus.

Sheep which are subject to convulsive ergotism, are probably also subject to the gangrenous form. We have abstained from considering it in the symptomatology because we have not met with documents which authorize us to generalize the symptoms and to extend them to the ovine species.

The gangrenous form is the only one which has been observed with the bovine species; it remains local and is not complicated with the greater part of the general symptoms which may precede it with the other species of the domestic animals that have just been referred to. The appetite is preserved, the muzzle remains moist, and the expression of the eye is not changed. These signs of health often remain until the fatal termination of the disease. The circulatory lesion seems then to be alone in play; it is localized in the digital region of the posterior limbs (Randall), or extends to the metacarpal and metatarsal regions of the limbs of one side (Decoste). A slight swelling of these parts announces the beginning. The hair becomes dull, the skin is dried, hardened, and mummified as well as the parts immediately beneath it. The appetite is preserved, but the animals become thin; a few individuals take flesh after the loss of the gangrenous limbs and may be prepared for the butcher. Death in a condition of marasmus is the most common result. When the affection takes a relatively benign form the gangrene does not destroy the part; the digital region loses its elasticity, the points of the toes are elevated, grow to an unusual length, and the weight is supported on the back of the fetlock. In this situation the animals pass a miserable existence until the owner, from pity, sends them to the butcher (Randall). The more benign form seems to us to have a resemblance to convulsive ergotism; the position of the posterior limbs has perhaps for its orign a contraction of the extensors.

In the records of epidemics of ergotism there are found but few references to the disease in the horse, and these are of no use in tracing the symptomatology. We are then reduced to a recapitulation of experiments to the number of two, one made by Hertwig and the other by Parola.

Hertwig administered to a horse within twenty-four days 3,552 grams [about 7 pounds] of ergot of rye. The phenomena observed were slight colics, loss of appetite, which disappeared within a few hours, drowsiness, which also soon passed away, dilation of the pupils, slight spasmodic contractions of the muscles of the skin and diminished temperature of the surface of the body The pulsations of the arteries were retarded from 40 to 28 to the minute. The day following the administration of the last dose all abnormal phenomena had disappeared.

Parola experimented on a vigorous and lively mule affected with a nasal discharge. During six days he gave it, in addition to its ordinary ration, ergot of rye in progressive doses of from 20 to 64 grams [¾ to 2 ounces] a day. The first day, the pulse was from 56 to 58, with lowering of the external temperature. The second day, pulse 58, respiration difficult, tearful eyes, loss of appetite, dullness, beating of heart insensible. The third day, coldness of the skin, general tremors, difficult respiration, spasmodic trouble of the cardiac and arterial pulsations, absence of appetite, apathy, dullness, suppression of the nasal discharge, which returned after the use of irritating injections. From the fourth to the seventh day, development of these symptoms, unsteady and difficult walk, trembling, inclined to lie, painful swelling of the knees, the nasal discharge definitely arrested. After having taken in all 284 grams [0.6 pounds] of ergot, the mule, which had continually lost in flesh and liveliness, became insensible and was destroyed.

It may be concluded from these facts that the horse escapes the pernicious effects of ergot no more than other animals, and that, placed under favorable conditions, one of the two forms of the disease may be clearly manifested, as the result of long and continued use. Ergot is also a poison for insects; in Poland they kill flies by giving as a bait powder of ergot mixed with honey. Leeches, plunged into an infusion of ergot, perish instantly (Lorinser).

Anatomical characters.—Studied for centuries, ergotism presents, in regard to its pathological anatomy, lamentable deficiencies as well with mankind as with animals. Considering the variation of symptoms it is useless to insist that the anatomical lesions cannot in all cases be the same. Those which we are about to enumerate all belong to either artificial or experimental ergotism.

The *rigor mortis* is never excessive; the flabby muscles are softened, the bones engorged with blood, particularly near the articulations. The venous system is distended by a black, pitchy, semi-fluid blood; the arteries, sometimes empty, contain in other circumstances a red fluid blood. In the thoracic cavity the lungs are found hepatized in the posterior portion; the heart flabby, small or voluminous, contains fibrous coagula and a black, viscid, semi-fluid blood; in the left side of the heart, which is often empty, there is nothing found but fibrinous concretions. The mucous membrane of the small intestine is pale, yellowish, infiltrated, and softened, sometimes covered with red striæ or black points. These black points are compounds of fat and pigment (T. O. Heusinger). The mucous membrane of the large intestine shows hyperæmia, which is far from being constant. The proventriculus of the gallinaceans has a gray or a wine-red appearance; its mucous membrane is ulcerated or covered with granulations; the gizzard is black (Millet). Do not the granulations depend upon the ulceration of the pepsine glands? The cerebral envelopes, principally at the base, are congested, engorged with a black blood resembling that found in the veins; a section of the brain shows sometimes a very apparent punctation; in other cases, much more common, it is anæmic. Analagous changes are met with in the spinal cord.

The local disorders of gangrenous ergotism are those of dry gangrene (see *Gangrene*).

Physiological action.—The symptoms of gangrenous or convulsive ergotism, as a whole, indicate incontestably that the nervous system fills the principal rôle. The phenomena observed by Hoppe when he placed ergotine in contact with the isolated organs of the frog or the rabbit would remove all doubt if any could exist. Ergotine causes a marked stimulation of the heart, followed by a weakening and a retardation of its movements; the intestine contracts, but the contraction is not renewed after a second application; the blood vessels dilate; soon follows a contraction with congestion of the *vasa vasorum* which swell and cause the walls of the veins and arteries to become rigid; the sensitive nerves are partially paralyzed; later their sensibility is increased; a general intoxication congests the brain and spinal cord. This last effect is not constant; the autopsies show that though the envelopes of the cerebro-spinal axis are always congested, wholly or in part, the nervous centers themselves are quite often anæmic.

It follows from these results which are conformable to what is observed in ergotism that the primitive phenomena depend upon a sedation of the sensory nervous system, and this anæsthesia is succeeded by a hyperæsthesia and a retardation of the circulation. The exaltation of the sensibility has not always the same seat; this circumstance explains the variations of the symptoms and the predominant lesions of a functional apparatus. In all cases the hyperæsthesia excites reflex action, sometimes in the intestinal tube (colic, vomiting, and diarrhea); at other times in the voluntary muscles (contractions and convulsions); at still other times in the involuntary muscles of the vessels (gangrene). These reflex actions may be successive, simultaneous, or they are developed separately and remain separated during the whole course of the disease. With man, who can give an account of his subjective sensations, intense pain precedes the contraction; then follows, according to the intensity of the disease, convulsions varying from trembling to epileptic attacks. The over stimulation leads to exhaustion, which brings a calm in the sensitive system. Intolerable pains, which are more localized, also precede the dry gangrene; the patient feels in the part which becomes the seat of it a cold sensation; later, this is recognized by the thermometer and to the patient it seems glacial; the sensation is lost when the gangrenous effect is accomplished. The dilatation of the vessels, followed by a narrowing of their channels are phenomena which are connected with primitive anæsthesia, a secondary hyperæthesia, and with reflex action, which affect the vaso-motor nerves. The rigidity and congestion of the vascular tubes, the weakening of the contraction of the heart, retard the circulation in the extremities, favor the stagnation of the blood, and consequently necrosis, even if the reflex phenomena are not sufficiently intense to obliterate the channel of the afferent vessel and produce mortification by anæmia. What is produced in an intense manner at one or several extremities is repeated at all the periphery in gangrenous and convulsive ergotism; the lowering of the temperature has no other cause than the retardation of the circulation and the reflex muscular spasm. The cerebral phenomena are probably only secondary and are due to the congestion of the brain and its envelopes; the anæmia of this organ would also account for them. It is a law that local congestions go side by side with local anæmia; when ergotism becomes chronic, the anæmia is generalized, a common result in all diseases which are of long duration. The absence of exudations removes all idea of inflammation, and if hepatization of the posterior lobes of the lungs has been found, particularly with the gallinaceans, this lesion is neither constant nor general.

The medium dose of this agent for medicinal purposes is given by Tabourin (*Nouveau Traité de Nat. Med., etc., II, p.* 447) as follows: Cattle and horses, one-half to one ounce (16 to 32 grams); goats, sheep, and pigs, one to two drachms (4 to 8 grams); dogs and cats, one-half to one drachm (2 to 4 grams). Finley Dun says: As a parturient or styptic, for the mare or cow, one-half to one ounce; for sheep, swine, and bitches, about one drachm (Vet. Medicine, p. 212).

HISTORY OF ERGOTISM.

Wood states that epidemics of ergotism or chronic ergotic poisoning have been recorded from time to time since the days of Galen [130 to 200, A. D.] and of Cæsar [B. C., 100 to 44]. (Therap. Mat. Med. and Tox., 4th ed., p. 565.) There is much reason for doubt, however, in regard to the diagnosis of cases occurring before the tenth century.

Verheyen says that, "From the ninth to the thirteenth centuries several grave epidemics appeared in France. The first chroniclers who made mention of them, faithful to their traditional habit, confounded them under the generic denomination of *plague* (*peste*). In the tenth century these epidemics received a special name; they were called *ignis sacer*, *arsura*, *claudes seu pestis igniaria*. In the twelfth century the nomenclature was increased with the terms *ignis sancti Antonii*, *sancti Martialis*, *Beatæ Virginis*, *ignis invisibilis*, *seu infernalis*. All these expressions were used to designate one and the same affection, which was no other than ergotism.

The learned historian of the epidemics of *feu sacré* of the Middle Ages, Professor Fuchs (*Das heilige Feuer im Mittelalter*, Berlin, 1834), fixes the first invasion in the year 857. This explicit passage of the chronicle leaves no doubt in this regard. *Plaga magna vesicarum turgentium grassatur in populo et detestabili eos putredine consumsit, ita ut membra dissoluta ante mortem deciderunt.* (Pertz, 11, 230.) The epidemic of 590 (Greg. Tur., X, 30) that some authors refer to the *feu sacré*, does not appear to us to have presented the characters of this. Its course was extraordinarily rapid; it began with a slight headache, a forerunner of death (*ita ut modico quisquis ægrotus capitis dolore, pulsatus, animam funderet*). These morbid characters can no more be considered ergotism than the very vague statement that near Limoges several were consumed by the *feu celeste* with which some were burned in Tourraine (*nonnulli ab hoc igne sunt adusti*). At the same time a very fatal epizootic occurred which did not spare the deer. A great drought had destroyed the herbage; it followed rains and inundations, conditions favorable to the evolution of charbonnous diseases. A fact supporting this view, as well as the opinion of Fuchs, is that rye, which is an Asiatic plant, was only introduced into cultivation during the Middle Ages (Link). Admitting, what is supposed, that Europe is indebted for it to the invasion of the Huns, it is still very necessary to take into account the condition of this part of the world before concluding that at the end of the sixth century the new cereal had become generalized and had entered into the regular agricultural rotation of Gaul.

All the epidemics of *feu sacré* correspond to years which were characterized by a rigorous winter followed by a very rainy summer, causing a deficiency in the harvests, and bringing scarcity and famine. The epidemics began about the month of September or October and terminated in the spring, unless the atmospheric condition of the following year continued to be fatal to the products of the earth. No mention is made of great epidemics of *feu sacré* during years which were characterized by productiveness, but local epidemics are met with which were circumscribed within the limits where the center of ergotism still exists in our days, and which comprises *la Sologne, le Dauphiné, le Lyonnaise, la Lorraine, and l'Artois.*

If we take into account the conditions which concur in the evolution of *feu sacré,* it may be established, *a priori,* that the sanitary state of the domestic animals was not more favorable than that of the human species, and that epizootics should have been more frequent than they are mentioned by the chroniclers. Are indications found in their annals which authorize us to attribute these epizootics to the same source, that is, to the *feu sacré?* This question cannot be solved; more than that, the accounts themselves forbid an hypothesis relative to the form and nature of simultaneous diseases. It is sufficient to cite this passage from the chronicle of St. Bavon, referring to the year 1127: *Plaga divina Franciam effligit, ignis scilicet corpora crucians. Pestilentia maxima facta est animalium.* What was this plague of animals? Formulated in this manner it is impossible to conjecture. If in the presence of such profound obscurities we are permitted to hazard an opinion, we would say that in all probability the food of the animals did not consist of rye in these calamitous years when scarcity and famine were general, and when the cause of *feu sacré* was unknown. Removed from the toxic factor, they did not escape its powerful accessories, and the diseases which decimated the herds must have been other than ergotism.* Perhaps the first epidemic of *feu sacré* which ravaged Portugal in 1189 was an exception, and it may be necessary to attribute the concomitant epizootic to ergotism. It is thus described: *Hujus etiam tempore morbi nunquam, ante, visi ingruebant, ferventissimis intra viscera ardoribus, quibus homines quasi quadam rabie exagitabantur. Exorta etiam fames, frugibus tam vi tempestatis, quam verminibus corruptis, et lues non minus nocens pecori quam hominibus et multorum relictæ vacuæ possessionibus* (C. F. Heusinger, Fuchs).

The uncertainty relative to the form and nature of the epizootics is one of the most common facts in the records of the Middle Age. Thus the beginning of the famous black plague in 1347 was preceded in several countries by epizootics no less fatal. *In primis hæc acerba pestis in brutis animalibus incohavit; scabies et lepræ totaliter apprimebant equos, boves, pecudes, et capras; ita ut pili de dorsi ipsorum depilabantur et cadebant et efficiebantur macri et debiles, et post paucos dies moriebantur. Deinde incipit hæc rabiosa pestis per universum mundum discurrendo in miserabiles homines lethaliter sævire* (Cutteis). This passage evidently refers to a gangrenous affection, which has nothing in common with *feu sacré,* for with the exception of the epidemic of Brittany, which occurred at the beginning of the black plague, no traces of ergotism are found until 1373, when it appeared anew in France (Tessier).

During the whole period of the Middle Age but rare epidemics of *feu sacré* are mentioned in the north of Europe. Heusinger believes that those of the fifteenth and sixteenth centuries, designated by the name of scorbutus, belonged to ergotism. This opinion appears to us to have foundation; in fact the celebrated botanist, Dodonæus, described the epidemic of scorbutus, which occurred in Belgium in 1556, and was characterized by gangrene of various parts of the body. He attributed it to the spoiled grain which was imported from Prussia, and says in another work (*Historia frumentorum, Anto.,* 1569), that bread made from spoiled rye (*seigle altéré*) causes the disease called by the Germans scorbutus. The gangrenous form of this affection, which did not conform exactly to that observed in France and Spain, was replaced in the course of the sixteenth century by the convulsive form. When the epidemics of ergotism of the South and those of the North of Europe are compared a very remarkable fact presents itself; in the South the gangrenous form is the rule; a few of the epidemics were complicated by muscular contractions (*contractures*); in the North, on the contrary, dry gangrene was a rather rare affection. This phenomenon is applicable to animals with which ergotism has been observed during the continuance of an epidemic as well as with those experimented upon. Fowls are the only exception, and with these gangrene of the comb is an almost constant result. Do the properties of ergot of rye differ in the north and in the south? Chemical analyses may, perhaps, inform us; in waiting for these we may admit that the phenomena are not dependent upon the dose. In 1840, after threshing, ergot made up half the harvest of Finland. The epidemic showed itself as acute febrile, convulsive ergotism. Death very often occurred within forty-eight hours (Haartman).

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* The author appears to have overlooked the fact that such a disease might have been produced by ergotized grasses.—D. E. S.

Dating from the seventeenth century observers were more attentive to the phenomena presented by animals during the epidemics of ergotism, but they notice the fact with a briefness which cannot satisfy the science of pathology, because the proper elements are not furnished for retracing the medical history. Brunner, the recorder of the epidemic of 1694, in *le Harz*, limits himself to this phrase: *Novi pecora, armenta, sues, equos, anseres quoque non fuisse a contagione immunia.* In spite of the contagion which he admits, Brunner leaves nothing equivocal in regard to the cause, for he says: *Degeneravit quoque secale et loco granorum alimentariorum protrusit cornicula nigra.* The *quoque* relates to oats which had equally undergone degeneration, the characters of which are not indicated; its meal produced vertigo in the persons who consumed it. It would have been interesting to indicate the effect produced in horses, but the author maintains an absolute silence in regard to this. Though distinguished botanists affirm that the oat is subject to ergot, we must admit that our researches to discover a fact where the injurious properties of ergoted oats have been noticed with animals have remained without success.

In the description of the epidemic in Silesia in 1722, we read that the King of Prussia ordered an exchange for sound rye of that affected with ergot, which, as usual, caused sickness of the horses and hogs (Hecker). Convulsive ergotism reappeared in Silesia and Bohemia in 1736. Antoine Soring, the historian, makes the remark that it is known and demonstrated by experiment that ergoted rye produces disease with fowls and mammals, and that when animals suffer during the epidemics of ergotism it is conclusive of the quantity and violent action of the ergot in the rye.

From 1765 to 1769 ergot was very abundant in Sweden in the rye and barley. The epidemics which followed were attributed by Linnæus to the grain of the *Raphanus raphanistrum*, from which is derived the name Raphania, which in Scandanavia is still given to convulsive ergotism. Wahlin, after having experimentally demonstrated the inocuousness of the seeds of Raphanus, observes that there is no reason for not accusing ergot when, in the course of an epidemic, domestic animals such as fowls and hogs present similar symptoms to what are seen in man. This passage tends to prove not only that the domestic animals contract convulsive ergotism, but also that the ergot of barley is as dangerous as that of rye. This is, besides, confirmed by Retzius when he asserts that beer brewed with ergoted barley becomes a cause of convulsive ergotism for those who consume it. In Hesse, it has been often observed, notably in 1770, that the heads of barley contained as many, if not more, long, black grains as the rye. In our times (1856) this same remark has been made by T. O. Heusinger.

Traube, who left a much esteemed description of the epidemic of 1770, which was very extensive in Hanover, says that so far as he was able to observe the facts for himself, he saw in the circle which he traversed a single pig attacked with convulsive ergotism. Horses eating the ergoted bread were not incommoded; bovine animals consumed the flour with repugnance, but also without inconvenience. Dogs and sheep were not affected, with the exception of the little village of Lohe, where 7 sheep succumbed after presenting the phenomena of the convulsive disease. These animals had pastured on the rye fields after the harvest, which was made in a very dry time, when an abundance was lost by shelling. Traube did not discover a single case of abortion which Soring and others thought they observed with hogs. One fact impressed him: traveling through the villages which still contained the sufferers, the following spring, he heard general complaints in regard to the small number of the young fowls. But few eggs were produced, and the hens did not sit. Nothing of the kind occurred in the villages which had been exempt from ergotism. Two fowls were sent him which presented the spasmodic symptoms; these birds, placed upon their feet, fell to one side, allowed the head to hang, and agitated the limbs. When they arose of themselves the phalanges were contracted spasmodically; they lived four weeks; no autopsy was made (*Geschichte der Kriebelkrankheit*, 1782, pp. 13 and 15).

It is seen that in these epidemics the convulsive form predominates, while in France the gangrenous form is almost exclusive. The last extensive epidemic from which this country suffered, occurred in 1750, and the ravages at this time recalled those of the Middle Age. It commenced in Sologne, its traditional center, and extended through Landes, Flanders, and Artois. The ergot formed a third of the threshed rye; animals which were fed upon it contracted the same gangrenous accidents as man (Salerne).

Toward the last third of the eighteenth century the epidemics of ergotism were no longer so frequent; the perfection of agriculture may claim a part in this happy result, but the greater part is incontestably due to the generalization of the culture of the potato in the North and of maize in the South. In spite of these alimentary guarantees ergotism was not extinguished. In the present century it was mentioned by Courhaut and Bordot, in 1855; Barrier observed the gangrenous form in the departments of Isère, Loire, Haute-Loire, Ardèche, and Rhone. Ergotism has reappeared in Russia, Finland, Sweden, and some cantons of Germany. In the epidemic described by Wagner (1831) the hogs which ate ergoted rye presented the same symptoms of the disease as the human species, and Helm saw in Pomerania 12 hogs which, a few hours after having consumed a ration of rye mixed with ergot, were taken with vertigo and convulsions. They moaned and uttered anxious cries; the posterior parts were paralyzed, and the animals manifested their sufferings by singular contortions. The last epidemic occurred in 1855; it appeared in Hesse, and concurred with that mentioned in France by Barrier.

A peculiarity worthy of remark was connected with the Hessian epidemic; the younger Heusinger, who recorded it, says that his father, professor at the University of Narbourg, who was charged by the Government to examine the harvests of the year, accomplished his mission before the threshing. In the sheaves of the cereal he found a large quantity of *Bromus secalinus* [common chess or cheat] rich in ergot, though the heads of the rye were exempt from it; and as this ergot presents all the physical characters belonging to that of rye, it becomes certain that this cereal is not always to be blamed as much as has been generally believed. Rye harvested on lands badly cultivated was infested with *Bromus;* when properly cultivated but little was produced. This fact demonstrates the great influence of agricultural progress on the extension of ergotism and its cause. In countries where agriculture is in an advanced condition, as in Belgium, ergotism, either in the gangrenous or convulsive form, is unknown. The observation of Heusinger is not the only one; in two communes of the principality of Waldeck the ergot of chess also caused an epidemic (Rœrig).

This fact is not without interest for the veterinarian, since straw makes up part of the food of the domesticated herbivora, and the plants mixed in the sheaves, with the nature of their productions, merits more attention than is generally bestowed upon it. During the continuance of the epidemic in Hesse, T. O. Heusinger collected information in regard to the diseases which affected domestic animals. He learned that in the commune of Roda, where the most people suffered, and where convulsive ergotism was most violent, the sheep presented symptoms which could be referred to poisoning by ergot with the more reason as these animals were fed with rye straw and received the screenings of the grain. The inhabitants complained of the great mortality among the sheep; the shepherds reported that several had jumped the inclosures of the pastures, that they were then taken with convulsions and turning in a circle had dropped dead as if thunderstricken. Abortions were frequent, as also early parturitions; the greater part of the lambs died.

Doctor Randall reports that in the State of New York a disease appears each winter among the cattle, which begins by a slight swelling of the lower parts of the posterior limbs, with stiffness of the joints. This affection, which has the appearance of being very mild, invariably terminates by dry gangrene of the parts first involved, which freeze after the mortification. In the severe climate of New York the animals winter in the fields, and the farmers attribute the disease to freezing. Randall observes that if this were the real cause a circular line of demarkation would not divide the dead and living parts as regularly as happens in this disease, and, finally, that the external appendages, less protected against the cold than the limbs when lying, should freeze sooner. He adds, and it is the opinion of several other physicians, that the affection is no other than gangrenous ergotism. Indeed the *Poa pratensis* is rich in ergot, and as it does not produce each year an equal quantity, Randall thinks that the cases more or less frequent correspond to the abundance of ergot. (Veterinarian, 1842.)

If, in presence of the facts enumerated, we cannot fail to recognize the existence of gangrenous and convulsive ergotism with animals, we must also admit that these facts are neither so precise nor have the rigorous correlation of cause and effect which is desirable in pathology; they do not even give the elements for a symptomatic table. Randall furnishes in this connection some important information; it agrees with that contained in the interesting observation of Decoste. (Rec., 1848.) These materials joined to the phenomena studied with animals in experiments permit us to trace the symptoms of gangrenous and convulsive ergotism.

Mr. Fleming, in his work entitled Animal Plagues, has compiled a considerable number of references to epidemics and epizootics of ergotism, which, while they contain a large part of the early records relating to this interesting subject, also illustrate the difficulty in deciding at the present time in regard to the real nature of some of the diseases to which allusions are made.

In A. D. 992 there was a long and severe winter and an extremely dry summer, followed by famine. The wheat crops were affected with blight or ergot, and the forage was generally of a bad quality. Soon after there was a widespread and deadly epidemic of ergotism (*feu sacré*) in France.

Ergotism was again prevalent in France in 994. (An. Pl., i, p. 58.)

In 1041 there was most unpropitious weather, accompanied by earthquakes, tempests, and inundations. It snowed heavily during harvest time. In many parts of Europe there were heavy rains throughout the year. Flanders was inundated by the sea, and there were great storms. The consequences of these disturbances were famine and disease in England, Germany, and France. Cattle and men appear to have suffered equally. "The plague of Divine Fire (*ignis divina*, ergotism, or erysipelas) afflicted many, who were saved only through the merits of the Blessed Virgin. And in all that year it was very sad in many and various things, both in tempests and in earth's fruits. And so much cattle perished in this year as no man before remembered, both through various diseases and through bad weather." (An. Pl., i, pp. 60, 61.)

For 1085 Mr. Fleming makes the following record: Epidemic erysipelas (ergotism?) in France, with inundations and famine. "In the year 1085 there was disease in plants, and also in animals, throughout the world." (An. Pl., i, p. 63.)

In 1089–'91, on the Continent, "in these years many men were killed by the *ignis sacer* (ergotism or gangrenous erysipelas), which destroyed their vitals, putrified their flesh, and blackened their limbs like to charcoal. Even if their lives were preserved their extremities were so affected that they were only reserved for a most pitiable existence." This epidemy is mentioned by several ancient chroniclers. Animals suffered as well as the human species. (An. Pl., i, p. 64.)

In 1099 gangrenous erysipelas (ergotism?) in France in the human species. From the severity of the epidemy, we may infer that animals also suffered. There were great inundations in England by the sea and by the rivers, whereby people, cattle, and whole towns were drowned. (An. Pl., i, p. 65.)

In 1127 the "divine plague" (ergotism?) appeared in mankind in France. Prayers to the Virgin Mary healed the afflicted, it is recorded. Great pestilence among animals.

In 1213 gangrenous erysipelas (*feu sacré*) in mankind in France and Spain.

> Neither was the scarcity limited to the fruits of the earth, nor disease to the human species, for birds, cattle, and sheep became sterile, and brought forth no young, and many riding and other horses perished for lack of straw and barley. (An. Pl., i, p. 71.)

In 1598, after inundations and heavy fogs, there was a general epizooty among cattle in Germany. In the same year there appeared ergotism in the human species. (An. Pl., i, p. 138.)

In 1694 an eruption of Mount Vesuvius. A supposed epidemy and epizooty of ergotism. Brunner writes:

> By what unfortunate combination of circumstances, for so many years, the whole of nature seemed to labor under an unhealthy atmosphere remains a secret. Many men, and those most learned, have written on the state of the air, and I have been a spectator of most grievous calamities; for not only did unwonted fevers attack and kill the human race, and would submit to no remedies, but also the beasts were harrassed by deadly diseases. I know that sheep, cattle, pigs, horses, and geese were not free from the contagion. There was also a lack of corn, not only on account of the inordinate consumption of it by the soldiers, but also from the character of the ground. Some of the corn was so plainly diseased that it was dangerous for man to eat of it. I know also that pease, which formed a great part of the food of the army, were infested and diseased by a small insect, which made a minute hole in them. I never remember seeing such an abundant crop of darnel (tares) mixed with the oats, and which prevented the making of good oat meal, our chief food, for it was needless

to attempt to labor on it, it was so bad. All grain disappeared, and in its place small, black, horn-shaped masses became apparent, which were highly injurious to mankind. These were named "St. Martin's corn." A woman was shown to me by a surgeon who suffered from convulsions every eleventh day, solely from eating this corrupt grain, and the same surgeon told me he had amputated a leg mortified from the same cause. (An. Pl., i, pp. 166–167.)

In 1721 the winter was mild, but the spring time cold and damp, and the remainder of the year wet. Locusts in France and the whole of Italy. Epidemic ergotism in Silesia during this and the next year, and scarlatina in man at St. Petersburg, Courland, and Lithuania. So notorious was it that diseased grain produced formidable diseases in the lower animals, that while the epidemic continued in Silesia the King of Prussia issued an edict forbidding the use of rye tainted by the ergot, because it seriously affected horses and pigs. (An. Pl., i, p. 234.)

Another strange phenomenon was the generally laborious parturitions of the domestic animals at this period:

> The sheep in many places lambed with great difficulty, so that the shepherds were obliged to use force to deliver them. Among the cattle one hears of nothing particular beyond the fact that the breeding cows and ewes brought forth their young with great difficulty so that force was obliged to be used to assist them. At Strelitz three fine young cows died from this laborious parturition. They strained so violently that all their internal organs were protruded. (An. Pl., i, p. 235.)

In this connection Mr. Fleming gives the following quotation from Hecker:

> The uncertainty pertaining to the nature of epizootics of the Middle Ages, leaves us in doubt as to whether some of them might not belong to that class which have a common origin with many of the epidemics of mankind. *The ignis sacer*, *arsura*, *claudes seu pestis igniaria*, *ignis Sancti Antonii*, *Sancti Martialis*, *Beatæ Virginis*, *ignis invisibilis*, *seu infernalis*, &c., would all seem to be employed to denote the same affection, and which we have reason to believe was ergotism. It is only by chance, as it were, that wide-spread and fatal diseases among the lower animals are mentioned as occurring coincidently with these obscurely named epidemies, and when we read that the causes of their outbreak were unfavorable weather, which brought about a diseased condition of the crops and pastures we are only partially enlightened as to the nature of the affection.
>
> The scorbutus of the fifteenth and sixteenth centuries has been supposed, with much reason, I think, to have been ergotism, and up to this period it appears to have developed in a gangrenous form. At this time, however, it changed to the convulsive type, which it has chiefly maintained to the present. A curious feature in this disease is shown as it appears in the South and North of Europe. In the South, the gangrenous form is the rule; in the North the convulsive form is particularly marked, and very rarely the dry gangrene; while a few of the epidemies present both characters. The same peculiarity is observable in the phenomena of ergotism in the lower animals during the existence of an epidemy, and it has also been shown to exist by experimentation; the only exception would appear to be in the case of gallinaceous birds, in which gangrene of the crest or comb is the most constant phenomenon. It is not until the seventeenth or eighteenth centuries that we can with certainty find authors describing ergotism in the epizootic form in animals and from that time till now observers have been numerous. (Page 234.)

Convulsive ergotism appeared in mankind in Silesia and Bohemia (1736), and Antoine Soring, the historian of the epidemy, notices that it had been remarked, and the subject had been demonstrated by experiment, that spurred rye produces disease in fowls and mammiferous animals, and that when we know positively that animals are affected in this way during epidemies of ergotism, we may conclude that the rye is very rich in ergot, and its action very violent. (An. Pl., i, p. 262.)

In 1754 a very extensive epidemy of ergotism prevailed in France, nearly approaching in its ravages those of the Middle Ages. It began at Sologne, its traditional birthplace, and spread through the Llandes, Flanders, and Artois. The ergot was so abundant that it formed one-third of the rye. The animals fed on it contracted the same gangrenous diseases as afflicted the human species. (An. Pl., i, p. 384.)

The next outbreak referred to is described as follows:

> An epidemic of ergotism was also reported as occurring in many northern countries, caused by the wheat, rye, and corn having been diseased. It lasted during this and the next year, and animals seem to have suffered. Wagner described it as it appeared in the marshy districts of Saxony, the circle of Schlieben, and on the banks of the Elster: "A light frost destroyed the blossom on the vine and the rye in 1831. Each partially withered blossom of the rye crop, instead of a healthy seed, brought forth a spur of ergot about three-fourths of an inch long. * * * In some houses, where the disease raged most violently, grain was found consisting of two parts of diseased and one of bitter rye, vetch and a variety of other seeds. * * * Pigs ate ergotized rye (*Mutterkorn*), and suffered from its effects. Dogs, however, instinctively, avoided it; but when compelled by hunger to eat it, they exhibited symptoms of madness (*Tollwuth*). I believe that such food was partaken of here and there by dogs, and that it assisted in producing madness, as dogs and cats were so affected that no man ever remembers seeing so many mad as during the existence of the ergotism (*Kriebelkrankheit*) among the people. This unhealthy grain may have had something to do with the sickness among the lower animals which prevailed at this time, and which was ascribed to the choleraic influence, though its share must have been small." (An. Pl. ii, p. 172.)

Raphania in pigs was witnessed by Dr. Helm:

> Twelve pigs of various ages were fed with rye which contained much ergot. A few hours afterwards convulsions set in, with foaming at the mouth; the animals grunted and groaned most piteously; became paralyzed in the hinder extremities, and expressed their agony in the strangest contortions. At first I presumed the disease arose from the bite of a mad dog, but on opening the first animal that died I discovered the nature of the malady by finding in the stomach much ergoted rye. The jaws were so tightly closed that with great difficulty a purge of white hellebore was introduced, and that was followed by a dose of vinegar and buttermilk and repeated douches of very cold water. By these means seven of the animals were saved. The other five died in the course of a few days. (An. Pl. ii, p. 197.)

In our own country we have no compilation of the references to animal diseases which may have been made from time to time, and hence it is impossible for me to give a history of ergotism in this country. My attention, however, has been called to the following article in the Farmers' Cabinet, Vol. iii (1838–'39), p. 161, which shows not only that the disease has occurred heretofore, but that its cause was recognized:

> I have just seen a number of the "Farmer and Gardener" of Baltimore, dated 19th June last, which contains an article on the "Hoof-ail" of cattle, and copied from the *Genesee Farmer*, upon which I will make a few remarks.
>
> The writer of the article, John B. Bowers, dates from Ledyard, and ascribes the loss of the hoofs in three cows to their having been fed for eight or nine days on spear grass (I suppose our green grass, *poa viridis*) affected with ergot. The conjecture is well founded, as you may be assured by referring to the fifth volume of the Memoirs of the "Philadelphia Society for Promoting Agriculture," p. 196, where you will see a paper of mine on the subject, which I think will leave no room to doubt as to the cause of the disease. It is a curious fact that the ergot of rye, if ground into meal with sound rye, when made into bread and eaten produces mortification of the lower extremities in France. (See Memoirs of the same society, volume 3, appendix, p. 5.)
>
> JAMES MEASE.
>
> CHESTNUT STREET, *October* 6, 1838.

Dr. James Mease, the writer of the above, gives the following additional information on the same subject:

> In the year 1803, the late Joseph Cooper, of New Jersey, informed me that he had observed the hay made of the natural green grass, or spear-grass (*poa viridis*), growing on his fine meadows, on Petty's Island, made by banking out the Delaware, to be occasionally affected with a black spear, about one-fourth or half an inch in length, somewhat resembling the ergot in rye, and that cattle eating such hay became affected with a disease in their hoofs, causing them sometimes to drop off. He ascribed the morbid production in the grass to neglect in supplying it with water from the river, by means of sluices, during the dry season. Upon my mentioning the facts soon after to the late William Rush, of Philadelphia, an extensive grazier, he confirmed them from his own observations at Blooming Grove, near Gray's Court, in the State of New York, in the winter succeeding the very dry summer of the year 1793. The hay was the produce of a bog meadow; it is presumed, therefore, that it was made from the same natural grass that grew in the meadows of Joseph Cooper.

Some years after, Mr. W. T. Woodman, of Tredyffrin Township, Chester County, Pennsylyania, communicated to me an account, in the following letter, of a similar disease, and from a like cause, among his father's cattle:

"Having observed the remark in the *Port Folio* for May, 1815, in the review of the third volume of the Memoirs of the Philadelphia Agricultural Society, that, 'as yet, in America we have never heard of any human person falling a victim to the ergot, nor indeed is it satisfactorily ascertained that it has ever been injurious to our animals, I think proper to communicate to you an account of a disease which in 1802 or 1803 prevailed in this neighborhood among milk cows particularly, but which also affected other cattle and horses. You will perceive that it was analogous to the one supposed to be occasioned by ergot.

"For my part I am entirely ignorant of the cause, but still I am. unwilling to ascribe it to ergot (with which rye in this neighborhood is more or less affected every year), for this reason, that milk cows, which are never fed with rye by our farmers or butter-makers, exhibited more violent symptoms than oxen or horses.

"The farmers attributed the disease to a peculiar mildew, which sometimes affects the grass on the bottom meadows of a small stream, the basin of which is very extensive, and very luxuriant, and entirely appropriated to meadow land, and suffered to lie under natural grass. No timothy or other grass seeds have ever been sown on it, to my knowledge.

"The cattle affected did not appear to lose their appetite, and while they ate heartily of hay and other food became daily more and more lean, manifesting great uneasiness, occasioned most probably by violent itching. Their hair in many places fell off, or was rubbed off by the animal in striving to scratch itself. After these symptoms had continued for some time, one or both hind feet became sore and the hoofs loose, at which period the animals began to grow better. Others lost their hoofs and part of their legs. Three of my father's cows lost both of their hind feet, and some others in the neighborhood were equally as bad. The legs began by drying and growing smaller from the hoof to half way between the fetlock and the hock, at which point it appeared as if a string of twine were tied very tight round the leg. Above this part the flesh was to all appearance in perfect health; the lower part was hard, black, and offensive. When the lower part became quite dry, and little else than bone, it separated and fell off, after which the animals lived and ate heartily, hobbling along on the remaining stumps. They even began to grow fat. Their health seemed perfect. They would, no doubt, have lived long in this state, and were killed only from motives of compassion.

"One cow belonging to my father, which had lost only one of her hind feet, and that at the first joint above the foot, bore a very strong, vigorous calf, which lived and did well. The cow also afforded as much milk after as before her misfortune, and was pastured on the same grass to which her disease was attributed when in a state of hay.

"I think the disease was never known but one season. The first symptom of it was observable in February, and it reached its crisis about the middle of May. Should this communication lead to any further observations on the nature and cause of the disease I shall be much pleased, and they may be of great service to the agriculturist. Should the disease ever again make its appearance I shall be more particular in my observations.

"I remain, very respectfully,

"WM. T. WOODMAN.

"P. S.—It should be observed, that though we have every year more or less of the ergot, the quantity of it is never considerable. I think there is seldom more than one pint to a hundred bushels of rye.

"Different remedies were tried, but none of them afforded any relief."

"Being desirious to acertain whether the disease of the grass to which Mr. W. referred had grown in meadows that had been deprived of their usual supply of water, I addressed a letter to him in reference to this point, and received the following answer, under date of June 10, 1815:

"Your favor of the 30th ultimo came to hand the 4th instant. Since the receipt of it I have made numerous inquiries, for the purpose of obtaining additional information respecting the disease (of which I communicated an account), and on the season preceding its prevalence, &c; but I regret to inform you that farmers in general are so deficient in observation, and so entirely out of practice of recording facts, that I have not been able satisfactorily to ascertain whether the season in which the "injurious hay was made" was a dry one or not.

"However, my father informs me that, as nearly as he can recollect, about that period the ditch which conveyed water to his meadow became so filled with mud and accumulations of mud and other matter as to render the supply very imperfect. As a deficiency of water appears to be the cause of the unwholesome qualities of the hay, it is highly probable that the injurious hay was made during the season that water was wanting. But shortly after this time the death of my grandfather in a great measure excluded my father from the benefit of the water. The original plantation being divided into two farms, and that of my father lying farther down the stream, the water of the artificial course is exhausted before it reaches his land. It should, however, be remarked that since that period he mows his grass while it is very young, and before the seeds are touched by the "smutty affection." Indeed, the farmers generally in this neighborhood, since their cattle were affected with the disease, are careful to mow much earlier than they did formerly.

"I am strongly induced to believe that Mr. Cooper ascribes the disease to the proper cause, for I have been correctly informed that a load of the injurious hay was sold to —— Rogers, who at that time kept the Buck Tavern, in second street, whose cow, in consequence of feeding on it, was affected with a disease of a similar nature.

Your friend, &c.,

W. T. WOODMAN"

The disease prevailed to a great extent in Orange County, New York, in the year 1820, and is very well and minutely described by Dr. Arnell, corresponding secretary of the agricultural society of that county. The facts detailed by him leave no doubt of the deaths of numerous cattle in his vicinity being caused by their eating hay made from some grass that was affected with the species of ergot observed in the produce of the meadows before mentioned, for he expressly mentions that the spear grass grew in the meadows in the towns of Wallkill and Blooming Grove, where the disease prevailed, and in a bog meadow soil. Dr. A. remarks that, "the hay was cut in June or beginning of July, immediately before harvest; that only cattle in good condition suffered from eating the diseased hay, the poor and meager escaping." The means of prevention pointed out by Dr. Arnell are similar to that judiciously recommended by Mr. Woodman, viz., to cut the grass early, before the ergot forms; or, if it be found in the grass, to defer cutting it until late, when experience proves that it may be safely used; for Dr. A. remarks that "the ergot then becomes dry and shriveled, without any of the flour or vegeto-animal matter which it usually contains." But the hay made from such late-made grass must be of little value, because Dr. A. says truly that "this spear grass is so early that if left to stand till the usual time of mowing meadows it loses all its succulent and nutritious properties." This agrees with our experience with respect to the spear grass of Pennsylvania, where it ripens next in order to the early *Anthroxanthum odoratum*, or sweet-scented meadow grass. Still, however, it may be useful by answering one purpose of food in all animals: to stimulate by distention, and to add to the stock of barn-yard manure. The various remedies tried to cure the disease in New York are enumerated by Dr. Arnell. Those that succeeded were:

1. Poultices of soap, rye-meal, and salt, to the legs and feet.
2. A wash of beef-brine, composed of saltpetre, and common salt, applied several times a day, and after washing and rubbing the feet with the bitter-sweet ointment. Of the animals thus treated, one only lost its hoof.

In the treatment of the disease, the first object to be attended to is to remove the cause producing it. This is to be effected by drenches of castor-oil, or sweet-oil and molasses and water warmed, to which may be added, if found necessary, after the failure to operate of the first dose, half a pound of glauber salts, dissolved in warm water. During the operation of the drench corn meal, rye, or oatmeal, mixed with a large proportion of warm water, and a handful of common salt to every bucket of it, should be freely given. The use of hay free from ergot is as obviously indispensable. A handful of salt should be given every day to promote digestion and give tone to the system. The local applications must be of a stimulating nature to rouse the activity of the circulation and of the absorbents, and to enable the part laboring under a deficiency of vital energy to resume its healthy functions, or to throw off the disease. Fish or beef-brine will answer as well as any, but they should be well rubbed on the feet and legs, for friction greatly tends to assist in restoring the health of the parts. To prevent the appearance of ergot in the grass, care should be taken, when practicable, to supply the meadows with water in dry seasons.

In the Genesee Farmer, 1857, p. 50, was published the following letter, clearly referring to this disease:

> Perhaps you are aware there is a complaint among cattle occasionally, in this part of the world, and it may be in many others. I have heard of it in Canada. I do not know the correct name. It is not the hoof-ail, although it attacks the hind feet of cattle, and, if not arrested, the limb will rot off, up to the second joint of the leg, and the animal must be killed, or it will die; after it has proceeded so far as to be incurable, the only way is to knock it in the head.
>
> I write this to inform your numerous readers of a cure we have here, although perhaps the remedy is generally known. It is to cut off the toes of the hind foot (in which only it appears), about an inch horizontal, so as to open the foot sufficiently there for the blood to come out; then put the foot in a stocking with plenty of tar at the toe. If taken in time this will effect a cure. It must be done early, however, when the animal first shows symptoms of the complaint, by a frequent and slight kicking out of the hind foot, as if pricked with something.
>
> I have heard the cause attributed to poisonous hay, such as smut. Do you or any of your correspondents know anything about it? If so, let us have your, or their, experience.
>
> CHILTON FORD.
>
> MORRISTOWN, *Saint Lawrence County, N. Y.*

Again, in the same periodical, 1857, p. 245, we find the following report of this trouble in Portage County, Ohio:

> In consequence of the appearance of a severe and fatal disease among cattle in some parts of Portage County (Ohio) the past winter, the Farmers' Association of Edinburg appointed the undersigned a committee to investigate the subject, and ascertain if possible the nature, cause, and cure of this malady. The report of this committee we herewith forward for publication in the Genesee Farmer, together with a resolution adopted by the association at the close of an instructive discussion upon the adoption of the report.
>
> *Report.*—The disease is not caused by freezing, neither is it what is called hoof-ail, foot-rot, or fouls. Its symptoms seem to be a deadness of the end of the tail, extending upward till in some cases the flesh separates from the bone and falls off. About the same time there is a purple appearance just at the edge of the hair, above the hoof. It then commences swelling, becomes feverish, extending upward to the ankle, and in some instances causing a separation of the coffin-bone from the pastern joint. The lameness is confined entirely to the hind feet. The blood is pale and thin, and in most cases the animal retains a good appetite till near the last. The cause we apprehend to be feeding on hay containing ergot (a parasitic fungus growing within the glumes of various grasses) in considerable quantities. We arrive at this conclusion from the fact that the hay fed by one individual who had lost a large number of cows contained much of this article, and also that the farmer from whom he purchased the hay lost cattle from the same disease, and in both instances cattle fed on other hay were not affected. In every well marked case of this disease it has been ascertained that the hay on which the animals were fed contained the ergot. The hay in which the ergot was found the most, was the kind called the June, or spear-grass, growing in old meadows where the soil is rich and the growth rank.
>
> The severe frost on the 31st of May 1856, is supposed by some to have been the cause of this disease in the grass by destroying the vitality of the seed before it arrived at perfection; while by others it is attributed to the extreme warm growing weather in June causing an overflow of sap.
>
> Although we consider the whole subject involved in much obscurity and uncertainty, and requiring further investigation, yet we are satisfied the best manner of treating the disease is immediate resort to restoratives and a change of diet, whereby an increase of animal heat and vitality is obtained, and at the same time making an application of suitable remedies to the affected parts, by cutting off the toes until they bleed, and blue vitriol moderately applied to the foot has in several instances been found beneficial. A free use of charcoal and salt in various ways is undoubtedly a good preventive; and a careful examination of the hay or grass on which stock is fed is indispensable; if ergot is found in hay it may be removed by threshing or tramping.
>
> Of the specific nature and properties of the ergot in hay, or whether they are identical

with that of rye, we are not well informed. The immediate effects of the latter in large doses is well known, but it has no affinity to the ordinary known effects of vegetable poisons. What effect would be produced by its gradual and continual use we are not in possession of sufficient information to warrant us in speaking positively; but we do suppose, after a careful examination, that it operates on the blood of the animal, and unless immediate remedies are applied it proves fatal.

P. BARRON, M. D.,
R. M. HART, Esq.,
J. Y. PEARSON,
JONAS BOND,
Committee.

The following resolution was unanimously adopted:

Resolved (inasmuch as the evidence adduced is conclusive), that ergot in hay is the cause of this disease. The association cannot decide that it is the real cause of a poison being introduced into the system, owing to our inability to analyze this substance; therefore we desire to ask the editors of our agricultural papers for more information, and to obtain a chemical analysis of ergot.

In the Chicago Tribune, March 14, 1884, appeared a letter signed J. Hosmer, containing the following paragraph, which, while it refers the disease to a different cause, evidently describes the same affection:

In 1873-'74, in Chariton County, Missouri, the winter was very severe, the mercury going to more than 20° below zero. The people on the open prairie, mostly Germans who had recently moved there, seeing that the native Missourians provided no shelter for their stock provided none themselves. In the spring from one to three in a flock of eight to ten had the "black leg." It commenced to separate just where the skin joins the hoof. The animal being in great pain, lapped the infected part, and the poison was thus transferred to the mouth. It was nothing more or less than gangrene, as the leg rotted and became putrid.

In the month of February, 1884, a letter written to the editor of the Breeder's Gazette by Cushman Brothers, of North East, Pa., in regard to a strange disease of dairy cattle there was referred to me. This letter, written February 19, and a second one from the same gentlemen, dated March 8, in reply to some inquiries of mine, contained the followlowing information:

The dairy in question contained 18 cows, and the disease appeared about January 1, 1884. The first indication was "cocked" ankles behind, the legs beginning to swell in a few hours, and in two days were "as large as the skin could hold." This swelling only extended as high as the hock joint. In about a week the hoofs began to come off; the parts beneath were red, but there was no formation of pus. The feet were apparently healthy between the claws, the appetite good, the eyes bright, and the cud regularly masticated. The animals had been kept in a warm stable all winter. At the latter date mentioned four had been killed, their appetite having finally failed, two more were very bad, "a scab having formed from top of hoof to several inches above ankle and leg rotting off;" the foot of one animal had come off at first joint above the hoof, the fore feet of none had been affected but with two the forelegs were then swelling above the knees. One that had been affected without losing hoofs and had apparently recovered, was taken down a second time. Many people said their feet were frozen, but the owner says they have now changed their minds. He had looked carefully in their mouths for sores but had found none.

This was undoubtedly ergotism, and was so diagnosed by Dr. Germer, the health officer of Erie, Pa., after the Kansas disease had been attributed to this cause.

In recent years several epizootics of ergotism have also been observed in New York by Professor Law, in Iowa by Professor Stalker, and in Colorado by Professor Faville; and it seems probable that when our veterinarians learn to recognize this disease and to refer it to its proper origin, we shall find that it is not an uncommon affection of cattle.

TREATMENT AND PREVENTION OF ERGOTISM.

When the first signs of this disorder appear the most important point to be attended to is to make a complete change of food, and to see that this is of good quality, nutritious, and free from ergot. It would also be proper to give a dose of physic (from one to two pounds of Epsom salts), in order to remove as much as possible of the poison still contained in the digestive organs, and to follow this with soft food, as mashes and roots. In the most severe cases, those in which a part of the limb is already lifeless, treatment will avail but little. The greater number of cases, however, have not advanced to this stage when lameness is first noticed, and these will be greatly benefited by removing the cause, and placing the animal under conditions favorable for resisting the poison. A very important condition is warmth. Even when animals are fed large quantities of ergot they seldom suffer except in cold weather; and consequently in attempting to check the advance of the disease, advantage should be taken of this fact by placing the cattle in warm sheds.

Another condition believed by some to have much influence on the development of ergotism is the water supply. With plenty of water always at hand it is believed that larger quantities of ergot may be taken for a longer time than when the water supply is deficient. In the cold winters which occur over much of our cattle-raising country, it is difficult to induce the animals to take a sufficient amount of water. Holes cut through the ice soon freeze over, and the weather is frequently so severe that the cattle will drink only a few swallows of water before they will leave to seek a shelter from the cutting winds, and when, later in the day, they try to obtain more water, the drinking holes are frozen over. Where ergotism prevails the watering should receive close attention.

Ergotism can probably be entirely prevented by cutting the hay before the seeds have formed. Both in Missouri and Illinois I saw the clearest examples of this. Hay composed of the same kinds of grass, cut upon the same land, was free from ergot or largely infested with it, according as it had been cut green or ripe. This matter is worthy of careful consideration. Hay cut green is more digestible and in every way more valuable than that which is allowed to become ripe and woody; and the latter is much more liable to produce severe disease, such as indigestion, impaction, and ergotism.

This fatal disorder may, therefore, be prevented in the future by proper and careful management; the chief points in this being to cut the hay before the seeds have formed, to see that the animals have a sufficient quantity of drinking water, to keep them in good condition by the liberal use of nutritious food, and to protect them as much as possible from the inclement weather. When it is found necessary to feed hay which contains a considerable quantity of ergot it is of course doubly important to look after the water supply and the shelter.

GEOGRAPHICAL DISTRIBUTION OF SOUTHERN CATTLE FEVER.

An approximately correct outline of the district permanently infected with southern fever is a matter of supreme importance, not only to the people who live within and near this district, but to those interested in live-stock in every part of the country. The losses which occur every summer, and which in some years have been really disastrous to the stock owners of certain sections, have been largely the result of ignorance of the districts from which it is dangerous to bring cattle in summer, and to which adult cattle cannot be taken with safety at any season of the year, unless they are to be slaughtered for beef within a short time after their arrival.

An attempt to make efficient laws to guard against this malady by regulating the movement of cattle from infected localities has generally failed to give relief, because these districts could not be accurately designated. States, therefore, as well as individuals, have been unable to protect themselves, and the losses have gone on year after year in spite of individual precautions and State laws. The cheap cattle of Georgia, Alabama, Mississippi, Arkansas, Southeastern Virginia, and other infected sections are at times carried to the most remote sections of the country, and when this is done in spring or summer extensive and fatal outbreaks of southern fever among the susceptible animals which cross their trail or mix with them upon their pastures is the general result.

Last year such outbreaks of disease are known to have occurred in New York, New Jersey, Pennsylvania, Maryland, Virginia, West Virginia, Ohio, Illinois, Missouri, Kansas, and even in Dakota. Colorado and Wyoming seem to have escaped, notwithstanding the introduction of Southern cattle, and this was supposed to be due to the peculiar climatic conditions, but the experience of 1844 shows that no such immunity exists, and that cattle are affected here as they are in other places.

It is evident now that this disease may be carried to all parts of the country, and that before anything can be accomplished toward preventing the very important losses which are now annually caused by it, we must have more accurate knowledge of the section from which cattle are liable to carry the infection. To obtain the information necessary to map out the infected district special agents have been employed, who have carefully traversed every county along the border-line of this district, and have investigated the cattle diseases with sufficient detail to locate the limits of the infected district in most counties with very great accuracy. In some counties a sharp line cannot be drawn, because it does not exist, but in such cases the line has been drawn sufficiently toward the uninfected district to embrace, as is believed, all the territory that was really infected at the time of examination. As this district is being continually enlarged by a gradual though very slow advance of the infection, it is not safe to buy cattle near this line for shipment to the North in summer, unless a careful personal investigation is made by the purchaser at the time.

The infected part of the South is clearly shown on the accompanying maps. To establish the boundary-line of this district beyond controversy our special agents have carefully reported the individual experience of thousands of farmers, and others who have moved cattle either northward or southward in the vicinity of this line. These reports are far too numerous and voluminous to publish in detail, and, consequently, a simple résumé of the facts as they are known to exist is all that we have attempted to give in this report. The location of the border-line of the infected district is as follows:

VIRGINIA.

The northeastern extremity of the line is in Accomack County on the Atlantic seaboard. The permanent existence of the disease on this peninsula has not heretofore been suspected by the country at large. A few facts had come to our notice within the past year or two which seemed to indicate that certain outbreaks of southern fever in Maryland had followed the introduction of cattle from Northampton County, which covers the extreme southern part of the peninsula. At the time it seemed impossible that this disease could have secured a permanent lodgement so far north, and the reports were scarcely credited.

A careful examination of the peninsula, however, has demonstrated the existence of the infection of southern cattle fever throughout Northampton County, and extending for 2 or 3 miles across the boundary into the southern part of Accomack County. The infection seems to have been in Northampton County so long that no one remembers a time when it was absent. It is said that there are local laws prohibiting the movement of cattle from Northampton to Accomack at any season of the year, but that recently they have not been strictly enforced. In 1880 a considerable number of cattle that had been running upon commons in Accomack County, 2 or 3 miles from the southern boundary, died with symptoms of southern fever. It was found by investigation that more or less cattle had died from pasturing on these commons every summer for the past ten years.

In April, 1881, a drove of about 50 head of cattle was collected in Northampton County and driven to market across Accomack. At Pungoteague a stop of several hours was made, and here at least six head of cattle contracted the disease during the following summer and died. Two miles farther north another halt was made for dinner, and in this vicinity nine of the native animals died. Twenty miles north of this the herd seems to have stopped again, and here a large number of native cattle died.

There seems to be no reason to doubt, then, that Northampton County has long been infected, and that the cattle from that section when driven among susceptible animals produce the same fatal results as has long been recognized to follow a similar movement of Texas and Gulf-coast cattle. The infected part of Accomack County is very narrow, perhaps not more than 5 miles wide, and it is said that the disease is more malignant toward the seacoast than it is in the parts which border on the bay. This is in harmony with the fact that southern fever is known to have existed along the seacoast in North Carolina and Virginia for many years before it invaded the interior.

A careful investigation of the counties north of the Rappahannock River failed to reveal any trace of the disease. Not only were all the cattle apparently in good health, but imported cattle had remained free from disease after their introduction. Several instances were related of bulls being brought from North or West and continuing to thrive in their new home. According to all the information attainable, then, there is no permanent infection north of the Rappahannock River.

Coming south of this river, we find that cattle brought from Gloucester and Matthews Counties to sections of the State farther north and west infect pastures and thus destroy native animals. The facts obtained indicate that Middlesex County has become entirely infected, but we were unable to obtain evidence of any permanent infection in Essex County. In King and Queen County the infection has reached the pastures in the vicinity of King and Queen Court House within the last four or five years, and it now survives there through the winter. Ten miles north of this cattle are susceptible to the disease and suffer when on the same pastures with others from south or east of that locality. While therefore the southeastern part of this county is certainly infected the greater part is still free. The same phenomena in regard to the extension of the disease that has been noticed in other parts of the South were also apparent here. The effects of the disease had been apparent for twenty years south and east of the court-house, but the pastures here had not become infected until about five years ago, and since then cattle have been frequently lost. In Caroline County there was an outbreak of disease in 1881 caused by cattle from Gloucester County.

In King William County we find the infection permanently located on the Pamunkey River 8 or 10 miles above the New Kent County line, where it seems to have been for the past twenty years.

In Hanover County the permanent infection has been at Hanover Court House and Ashland for a considerable number of years. It is well known to the inhabitants that cattle brought from north or west of these places are nearly certain to die either the first or second summer after their arrival. The infection has extended but little beyond these two points.

Henrico appears to be entirely overrun with the infection. All the cattle sold from this and the surrounding counties go to Richmond either to be killed for beef or to be shipped by boat to other markets, and consequently there is little opportunity to collect instances of disease caused by cattle carried from here to uninfected localities. On the other hand it is admitted pretty generally by those who handle cattle that it is very dangerous if not absolutely fatal to bring these from the elevated sections of the State to any part of this county.

In Goochland County there have been very few cases of disease for a long time, but the southeastern extremity of the county is recognized to be dangerous to northern cattle. Farms on the James River three or four miles above Goochland Court House lose a few animals from year to year with southern fever. There is, however, little opportunity for the disease to occur, since the traffic in cattle is not extensive. Animals are not brought from north or west because of their liability to become affected, and they are not brought from the South because this is believed to be dangerous to the natives.

Powhatan County is undoubtedly infected, and has been in this condition for many years. It is reported by all the farmers along the James River to be absolutely fatal to cattle to be brought from north of the river to the south side. They suffer to the same extent when simply carried from the north to the south bank as when the distance is greater. Cattle from this county have long been considered dangerous to the native stock with which they came in contact when being driven to localities farther north. The introduction of this disease is not remembered by the oldest inhabitants, but so far back as the memory of man goes the present conditions have prevailed.

Cumberland County does not appear quite so thoroughly infected. At Trenton Mills and McRea's there is no doubt of the permanent infection, but the southern extremity of the county still seems to be free from it. In the counties along the river it is very plain that the sections immediately adjacent to the river have been longer infected and that the disease here is more generally diffused and more virulent than in the same counties at a greater distance from the water. And accordingly as we attempt to investigate the condition of other sections we encounter the difficulty of finding a smaller number of cases and a greater uncertainty in the minds of the inhabitants as to whether the native pastures really hold the infection from year to year, or whether each outbreak is the result of a fresh importation.

In Buckingham County our reports confirm the statements that were made last year. The infection extends up the south bank of the James River to a point slightly beyond the confluence of the James and Slate Rivers. From here the boundary line of the infected district passes up along the west bank of the Slate to Diana Mills; then the direction is a southeastern one to the vicinity of Gravel Hill, and to McRea's, in Cumberland County.

In Amelia and Nottoway Counties it has been impossible to trace any line or even to demonstrate the complete infection of the territory. It is generally admitted that twenty years ago and longer there was a complete infection of this district, but of late years there has been much less disease and it has become possible to bring in cattle from north and west with safety. In these counties there are no fences, and each man must necessarily keep his cattle upon his own pastures; as a consequence there is none of the indiscriminate mixing of cattle which used to occur, and the chances of contagion are greatly lessened.

While there have been considerable losses in Prince Edward County there is little evidence of permanent infection, except, perhaps, in the extreme eastern part. Most of the cases seem to have been the result of pasturing on commons that had been frequented by animals from Lunenburg County.

In Lunenburg there is no doubt of permanent infection. It is dangerous to bring cattle from west or north into this county, and, on the other hand, cattle from this county have frequently spread disease when driven toward the west or north.

In Charlotte County the boundary of infection becomes plainer and follows very nearly the line of the Richmond and Danville Railroad in its whole course across the county.

From the point where this railroad crosses the Staunton River to the North Carolina boundary, the line of infection was definitely located in my last year's report. The accompanying map delineates the portion of Virginia permanently infected with southern cattle fever as correctly as this could possibly be done. Parts of the line have been retraced three and four times in order to have it satisfactorily located.

NORTH CAROLINA.

The Blue Ridge Mountains of North Carolina are now looked upon by the people of this State as the practical boundary line of the district permanently infected with southern fever. At some points, as in Wilkes County, the infection has not quite reached the mountains, and at one other point it has crossed the ridge and invaded Henderson County. The laws of the State, however, make the Blue Ridge the line, and prohibit the movement of cattle from the eastern counties at all seasons of the year.

SOUTH CAROLINA AND GEORGIA.

The whole territory of South Carolina seems to have been overrun with this infection. The Blue Ridge Mountains, which form a part of the northwestern boundary of the State, have here been crossed by this contagion and are no longer to be considered as the line. The infected district beyond these mountains is, however, at present of small extent, and the advance is so slow as to be scarcely appreciable.

The small portion of Georgia which has heretofore been considered free from this infection is being rapidly overrun; and it is now doubtful if any of even the northern tier of counties can be considered entirely free. The mountain sections are not so thoroughly infected, and it is probable that Towns, Union, and Fannin Counties are practically free from the permanent infection.

Whitfield and Murray Counties have been quite thoroughly inspected and the commons of both found to be infected in all parts. Many of the farms are also infected, but some still remain free, especially near the northern boundary. Cattle taken from these counties to the mountain ranges of Gilmer for pasture have not so far as has been ascertained caused any outbreaks of disease. This is probably due to the fact that Gilmer County is also pretty thoroughly overrun.

Here, as in many other localities, there are evidences of a different intensity of the contagion in various parts of the counties, but more particularly in different sections of the State. Thus, cattle which have pastured on the ordinary infected ranges of Whitfield County without harm, have become diseased as a consequence of feeding along the trails and on the commons where cattle from Southern Georgia had lately grazed. The movement of bovine animals from one farm to another or from one county to another is also considered dangerous. The fatigue induced by driving is without doubt one of the factors in producing the disease in such cases. Even cattle from the extreme south often succumb when exhausted by long journeys. Similar facts have long been noticed with other diseases, and particularly with anthrax, cattle which have resisted the contagion on the infected farms becoming victims to the virus already within their bodies, when their vital resistance is lowered by great fatigue.

At Dalton there is a probability that permanent infection existed before the war, but not to the same extent as at present. The cattle driven from South Georgia to provision the armies, and later those brought by the refugees returning to their homes, are believed to have been the means of distributing and intensifying the contagion throughout Northwest Georgia.

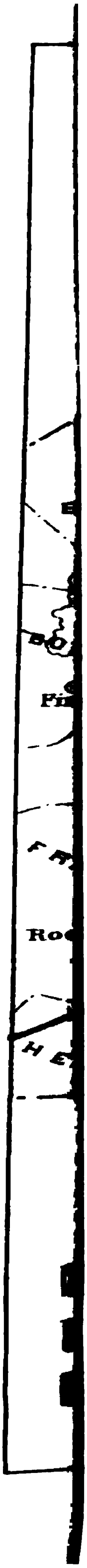
TENNESSEE.

The line of the infected district crosses the boundary line between Georgia and Tennessee near the western slope of the mountains, and follows a northwesterly direction to Parkville and Benton, in Polk County; then its direction is nearly directly west to Cleveland, Bradley County, and to Snow Hill and Harrison, in James County. From this point the river becomes the line across Hamilton County, Chattanooga and the Chickamauga Valley, having been infected for a long time. The southwestern part of Polk and the southern parts of Bradley, James, and Hamilton Counties are, therefore, permanently infected. The district infected is here, as we have found to be the case elsewhere, extending slowly toward the north; and though this extension is slow it is apparently continuous, and the territory once overrun is seldom redeemed from the scourge. The points which we have mentioned as existing on the border line of the district have only been infected within a few years, and, as elsewhere in newly infected sections, it is the commons rather than the farms that are dangerous, and even the commons are not uniformly affected.

In Marion County the line of infection passes in a northwesterly direction up the east side of the Sequatchie Valley and within one or two miles of the Sequatchie County line, and then crosses the valley and down the west side for five or ten miles, passing around the mountain range and taking a southwesterly direction to the vicinity of Jasper; from here the direction is again northwesterly to Decherd, in Franklin County, the greater part of this county being apparently permanently infected. From Decherd the direction of the line is slightly south of west to Fayetteville, in Lincoln County, and from this point along the Elk River to the Alabama State line in the vicinity of Veto Station. The southern part of Lincoln County is what is called the barren region, and has been infected for many years. In the vicinity of Marbut's the line again crosses into Tennessee, taking a northwestern direction, and at the county line between Giles and Lawrence Counties is about five miles north of the Alabama State line. On the boundary between Lawrence and Wayne Counties this line is 10 miles north of Alabama. At this point there is a sudden bend toward the north, the line crossing to the northwestern corner of Wayne County, taking in about one-fifth of Decatur County and all of Harden. In Henderson County it reaches as far north as Shady Hill, then passes directly west to the vicinity of Mifflin, in the same county. From here the direction is slightly south of west to Denmark, the boundary between Madison being crossed 2 miles north of Hardeman County; from here the direction is westerly to Stanton Depot, in Haywood County, and onward toward the southwestern corner of Haywood and along the northern boundary of Shelby to the Mississippi River. All of Shelby and Fayette Counties appear to be infected, while Tipton, as far as we have been able to learn, is free from any infection.

This concludes our examination of the district permanently infected with southern cattle fever for the year. It will be seen that a considerable portion of Tennessee has already become infected. Even the mountainous counties in the southeastern part of the State have been invaded, while in the river valleys of the central part of the State the line has reached considerably farther toward the north. Along the whole line of the infected district in this State, as in the other States previously examined, we have found the same history of the extension of the permanently-infected district. At some points this extension has been insignificant or is scarcely noticeable within the last quarter of a century, but in the situations more favorable to the progress of the disease there has apparently been a regular advance of from one to four miles per year. This history coincides substantially with what was learned of the progress of the disease in Virginia, North Carolina, and Georgia. As a consequence of these facts there can be no longer substantial reason to doubt the continued extension toward the north of the district permanently infected with this disease. Considering the extreme temperature which occurs in the mountains of southeastern Tennessee and in the part of this State located in the Mississippi Valley, we can scarcely hope that the winters in any considerable part of the stock-raising section of the country will be sufficiently severe to prove a permanent check to the extension of this contagion.

It is proposed to continue the examination of this district across Arkansas, Indian Territory, and Texas, to the Rio Grande River, and it is believed that a definite location of this line will be of great assistance to those engaged in the live-stock industry in the whole southwestern part of the country. The mortality among thoroughbred cattle taken south of the border line of the permanently infected district is so great that it has become a matter of importance to buy animals which have acquired a certain amount of immunity from this disease. It is believed by many breeders that by establishing breeding farms just within the line of infection that there will be a smaller mortality from the disease, and that the animals raised under these conditions will still be able to resist its effects in a very perfect manner. Already such farms have been established in Southeast Kansas and Southern Missouri, under the belief that animals raised in this locality will prove insusceptible to the disease when carried further south, but the great uncertainty which at present exists with regard to the exact location of this line makes it extremely doubtful if these farms have been correctly located. A number of extensive breeders who have a very intelligent idea of the nature and effects of this disease have recently expressed to me their high appre-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ciation of the work now being done by the Department of Agriculture toward establishing the boundary of this infected district. It is believed that definite knowledge in regard to this will relieve them from many of the causes of embarrassment connected with the shipment of thoroughbred cattle to the South.

INVESTIGATIONS OF SWINE-PLAGUE.

In a communication of M. Pasteur to the Paris Academy of Sciences (Comptes Rendus, 1883, p, 1163) it was asserted:

1. That the microbe of swine plague is a dumb-bell micrococcus.
2. That pigeons are very susceptible to the virus, and passing this through a succession of these birds increases its activity.
3. That rabbits are also susceptible, and passing the virus through a succession of these animals attenuates it to such an extent that if pigs are inoculated with it they only contract a slight illness which grants them immunity from subsequent attacks.

To these assertions Dr. Klein (Vet. Jr., 1884, July, p. 39) replies:

1. That M. Pasteur has overlooked the true microbe, and that this is a bacillus and not a micrococcus.
2. That all of his (Klein's) inoculations of pigeons with virus taken directly from diseased swine—virus which invariably produces the disease in swine and other susceptible animals—and with his artificial cultures of the organism of swine fever, produced absolutely no effect, either general or local.
3. That it is impossible to say whether M. Pasteur's rabbits died of swine fever or of septicæmia, though he (Klein) had shown in 1877 that rabbits are susceptible to swine fever when inoculated from material directly derived from the pig.
4. He adds in an addendum that he has recently satisfied himself that the artificial cultivation of the virus in the organs of mice or rabbits by inoculating these from diseased swine will produce a mild form of swine plague from which the animal quickly recovers, and is thereby protected from the disease.

It is very evident that before any safe method of protective inoculation or vaccination can be adopted we must be satisfied as to the nature of the virus. Rabbits and mice are both subject to septicæmia, and it is quite certain, from the difference in the microscopical appearance of the germ described by these two investigators, that either the one or the other had cultivated and inoculated with a septic virus. Dr. Klein does not hesitate to say that it seems probable to him that, "as in the case of the microbe of fowl cholera, M. Pasteur did not work with pure cultivations of the microbe of swine fever." M. Pasteur will doubtless say, on the other hand, that Dr. Klein has evidently been cultivating and inoculating with the septic vibrio. Both cannot be right in their belief that they have been working with the true germ, and, consequently, it is very probable that both sets of hogs were not protected from the genuine swine plague. Each has made many inoculation experiments, each has cultivated his germ through a number of cultivations in purity as he supposes, and each believes that he has produced the true swine plague with such cultivations; but one of them is wrong; vaccination with the virus of one will fail in practice, and if the wrong virus is so easily obtained it becomes doubly important to know how to discriminate between them.

In former reports I have given details of experiments which, if correctly stated, demonstrate beyond question that the microbe of swine plague is a micrococcus. These experiments were made and the accounts of them published in advance of those of M. Pasteur, and the evidence furnished was all that could reasonably be required to decide a scientific question of this kind. Dr. Klein, however, has published evidence which on its face is equally conclusive in his favor; and as it is not likely that two different diseases resembling each other so closely in symptoms and lesions, but having such dissimilar virus, have been investigated, the most reasonable conclusion is that one is mistaken in his conclusion. It is necessary, therefore, to review certain points in the investigations and to bring forward such new evidence as shall be required to remove these uncertainties.

1. *The microbe of swine plague.*—As I have shown elsewhere (*Science*, 1884, p. 155) Dr. Klein was first to demonstrate the presence of micrococci in the tissues of animals that had suffered from swine plague, but he did not at that time (1876) attribute, nor has he at any time subsequently attributed, the cause of the disease to this organism. On the contrary, he published a long series of investigations in 1878 (report of the medical officer of the Local Government Board) from which he concluded that the true germ of this disease is a bacillus, and in his last paper reiterates this conclusion and asserts that the micrococcus is entirely an epiphenomenon (Vet. Journal, July, 1884, p. 39–47).

In my report for 1880 (Department of Agriculture, Special Report No. 34, pp. 22–24), I published experiments showing that the blood of sick, not dead, hogs, which had been received into vacuum tubes that were thrust inside the vein with proper precautions before being opened, and were then immediately withdrawn and hermetically sealed, contained micrococci and no other organisms, and that hogs inoculated with this blood contracted a severe form of swine plague. This organism was found to exist in the virulent liquids (blood, peritoneal effusion, &c.), in three distinct outbreaks of the disease which were investigated at that time. This was the first discovery recorded, so far as I am aware, of the existence of micrococci in the blood of the affected swine before death; and it has a very important bearing on the etiology of the disease, since a *post mortem* development of the germs is out of the question, and they were found in situations to which there was no direct communication from the outside of the body.

In my next report (Department of Agriculture, Annual Report, 1881 and 1882, pp. 267–269) I gave the details of experiments which demonstrated that these micrococci after they had been carried through six cultivations in considerable quantities of liquid were still capable of producing very marked cases of the disease. This was, I believe, the first satisfactory evidence of the pathogenic effect of the micrococci in the disease known as swine plague; and I desire to call attention to the fact that these inoculations were made January 17, 1881, or more than fourteen months before the discovery of the same organism by MM. Pasteur and Thuillier.

To establish the connection of the bacilli with the cause of the disease, Dr. Klein relies upon the following evidence:

1. The presence of bacilli in microscopic sections of the tissues.
2. The multiplication of bacilli in the artificial cultures of the virus.
3. The production of disease by inoculations with the cultivated bacilli.

He has not forgotten that in his first report he described micrococci and not bacilli as existing in the tissues, but there is an evident attempt to explain this by conveying the impression that these were found exclusively in situations where they might be derived from external sources.

For instance, in his last paper (Vet. Journal, July, 1884, p. 41) he says:

> Preparing sections through the typically ulcerated mucous membrane of the large intestine, staining these in aniline dyes, and examining them under the microscope, I find this: In the superficial parts of the necrosed membrane are present large numbers of micrococci of various kinds, chiefly varying in the size of the elements and in the mode of aggregation. These micrococci stain well in Spiller's purple and in methyl blue, and are present only in the necrotic parts of the ulceration, in which they appear irregularly distributed. But in the depth of the tissue, and extending in many cases into the inflamed sub-mucous tissue, are seen streaks and clumps of minute rod-shaped bacteria, which coincide as regards size (length and thickness) with the bacilli which I described in my former memoir, the single organisms being about 0.001 to 0.004 ^{mm}. long, and about a third or a fourth as thick.

In his first report he described the situation of the micrococci in the intestine somewhat differently, as follows:

> From, and even before the first signs of necrosis of the mucosa, viz., when the epithelium begins to break down and be shed from the surface, there are found masses of micrococci, which in some ulcers occupy a great portion of débris. (Report of the Medical Officer of the Privy Council and Local Government Board, 1876, p. 98.)

Again, in regard to the ulcerations of the mucous membrane of the tongue, he says in his last report:

> I have seen in the superficial parts of the ulcers large clumps of micrococci, but in the depth of, and extending between the inflamed muscular tissue I have found the same rod-shaped organisms as mentioned above; they are chiefly in spaces between the bundles of the inflamed connective tissue, forming here streaks of longer or shorter chains. (Page 42.)

In his first report this was stated as follows:

> In the ulceration of the tongue just mentioned, and at a time when the superficial scab has not been removed, I have seen masses of micrococci situate chiefly in the tissue of the papillæ, but at some places reaching as far deep as the inflammation extends. (Page 99.)

In regard to the similar lesions of the epiglotis the following language was used:

> I have before me preparations through the epiglottis, the submucosa of the posterior surface being in a state of necrosis, and near the edge so broken down as to leave there a deep ulcer, while the mucous membrane of the anterior surface is only slightly inflamed in its submucous tissue; in this I find lymphatic vessels filled with micrococci, &c. (Page 100.)

As to the appearance of the lung his last report says:

> Sections through the diseased parts of the lung reveal, in preparations stained as above, the presence of large numbers of micrococci in the cavity of the bronchi and air vesicles, but not in all lungs, since I have found lungs in which they were altogether absent. But there are always present in larger or smaller clumps the same minute rod-shaped organisms as mentioned above. They are imbedded in a coagulum filling the air vesicles, or they block up a blood-vessel in the wall of a bronchiole or air vesicle. In the air vesicles I have seen exudation cells, white-blood corpuscles containing clumps of the rods; they are well brought out by Spiller's purple. In the air vesicles of some lungs I have seen them grow to very long chains, leptothrix, ten, twenty, and more times the length of the single rods. These rods were present, not only in the air vesicles, but also in the tissue itself, both of the walls of the air vesicles as well as of the smaller or larger bronchi. (Pages 41, 42.)

In his first report there is a most radical difference in the description of the situation where the micrococci were seen:

> In the infiltrated, firm, more or less disintegrating parts I find great masses of micrococci filling up capillaries and veins, and also contained in lymphatics around arteries. They may be found also in minor bronchi which have been completly blocked up by cheesy inflammatory products, but there the masses of micrococci, conspicuous by their blue coloration in hæmatoxylin preparations, are generally present in greater or smaller lumps between the outer surface of the plug and the wall of the bronchus.
>
> The pleura is much swollen, and contains great numbers, continuous layers, of lumps of micrococci. The free surface of the membrane is in many parts covered with them. The exudation fluid is also charged with them, as has been mentioned above. (Pages 100, 101.)

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by the use of enormous doses of virus that I have succeeded in reducing it to four or five days; and, therefore, when we are told that in these

SWINE-PLAGUE MICROCOCCUS
Photo-micrograph of cultivation liquid X 436.

That is to say, in 1876, Dr. Klein was able to find the micrococci not only in the necrotic parts of the ulcerations, but he found them *from and before the first signs of necrosis;* he found them *extending as deep into the tissue of the tongue as the inflammation extended*, and in the epiglottis *at a point where the submucous tissue was only slightly inflamed he found the lymphatic vessels filled with micrococci.* In the lungs, instead of the micrococci being confined to the cavity of the air vesicles and bronchi as he desires us to understand from his last report, he really found them *in the infiltrated and firm parts, filling up capillaries, veins and lymphatics.* They had even penetrated to the pleura *which contained great numbers and continuous layers of them; the free surface was covered with them, and the exudation fluid was charged with them.* Their presence in the pleural effusion is sufficient evidence that cross-section of bacilli had not been mistaken for micrococci in the tissues; and it may, consequently, be accepted as beyond question that this organism existed at the points named in the report of 1876.

In the last report it is stated that the rods (bacilli) are found "in the bronchial exudation, in the juice of the lung tissue, in the peritoneal exudation, and occasionally, but not generally, also in the blood already in the fresh state." Sections made through the fresh or hardened, swollen mesenteric and inguinal lymph glands are said to reveal the presence of clumps of the same minute rod-shaped organisms. Looking at a clump of these organisms, one imagines them at first to be a zooglœa of micrococci, but using oil-immersion lenses and Abbe's sub-stage condenser it becomes certain that they are undoubted rods—some smooth and uniform, others more or less "beaded."

In the results of the examination of the tissues it will be seen that, with the exception of the lymph glands mentioned, the bacilli of the last report have little if any advantage in situation over the micrococci of the first report. And if we consider that the organisms of these glands so closely resemble micrococci that it requires oil-immersion lenses and an Abbe condenser to make a distinction, and that even under such favorable conditions some of the rods are more or less "beaded," the reader will not feel so certain that they are undoubted rods as is Dr. Klein.

The examination of the tissues of mice and rabbits which have died after inoculation with the more or less septic liquid of dead hogs cannot be accepted as throwing any satisfactory light on so difficult a problem, since others cannot fail to have the same doubts in regard to Dr. Klein's experimental animals that this gentleman is so free to express in regard to those of M. Pasteur. The question as to the organisms found in the tissues of animals so susceptible to various forms of septicæmia as mice and rabbits after they have been inoculated with morbid products from hogs which have died of a disease in which local necrosis and gangrene is not uncommon, is one which can only complicate the real issue without in any sense elucidating it. Indeed, when Dr. Klein tells us that he has "seen a good many pigs inoculated with culture of the bacterium of swine fever, which beyond the swelling of the glands and beyond a transitory rise of the body temperature on the second and third day, by one or even two degrees C., showed no other signs," we have strong suspicions that the slight trouble produced was of a septic nature rather than a mild attack of the destructive swine plague. The period of incubation in swine plague is much longer than that of septicæmia; sometimes it is three weeks; generally it is from twelve days to two weeks, and it is only by the use of enormous doses of virus that I have succeeded in reducing it to four or five days; and, therefore, when we are told that in these

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Plate XI

SWINE-PLAGUE MICROCOCCUS

Photo-micrograph of cultivation liquid X 436.

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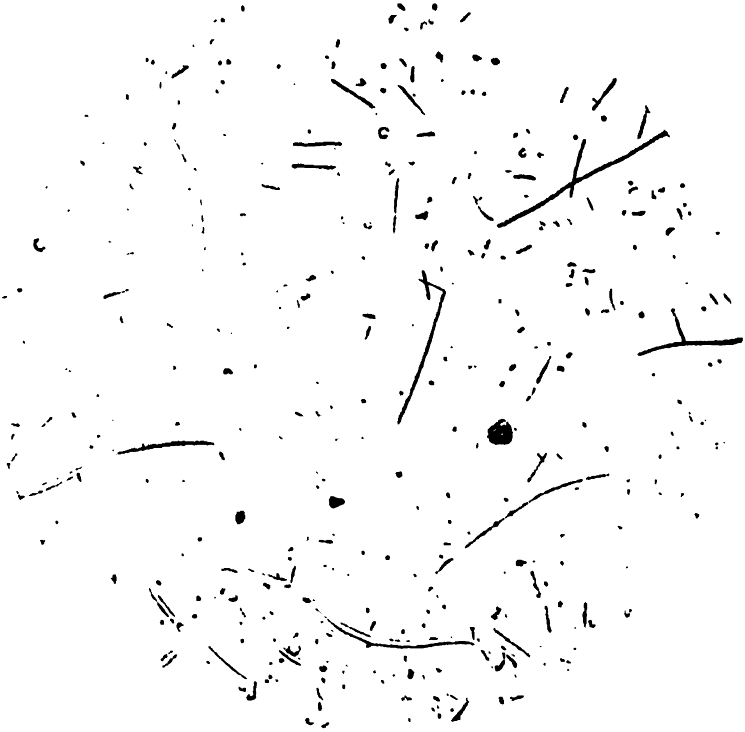

SEPTIC BACTERIA.
Photo-micrograph of Peritoneal Effusion.

A. Hoen & Co. [illegible]

mild attacks the period of incubation was but two or three days, and that in at least one case there was a rise of temperature within twenty-four hours (*Ibid.*, p. 43), the appearances are certainly very much more in favor of septicæmia than swine plague. Certain it is that in none of my numerous inoculation experiments has there been a rise of temperature within so short a time. As I write this I have just returned from making a *post mortem* examination of a pig killed in the last stages of the acute form of the disease; this was one of a lot of three inoculated with a virus so virulent that not one of a considerable number of swine that have been inoculated with it during the last three months has recovered. With so virulent a virus one would expect the incubation to be at its shortest duration, and yet neither of these three showed any appreciable signs of disease up to the twelfth day. All sickened at about the same time, and to-day, the fifteenth day, all were so extremely ill that the most careful prognosis would be death of all within forty-eight hours.

In animals which have died from the disease and on which a *post-mortem* examination was not possible immediately after death, I have also found bacilli in the peritoneal and plural effusion, and even in the blood. A photograph of some of the peritoneal effusion dried on a cover-glass at the time of the autopsy, and afterward stained and mounted, shows these very plainly; this photograph has been reproduced by the heliocaustic process and accompanies this report as Plate XII. No doubt bacilli would also have been found in the solid tissues of this animal; but these organisms were the result of changes which occur either shortly before or after death, and have not been found in any of the numerous animals which I have destroyed for examination when in the earlier stages of the disease. In such cases the peritoneal, the pleural, and the pericardial effusions, and usually the blood, are found to contain motionless micrococci of the figure-of-eight form, but often united in chains and various-shaped clusters.

In the many cultivations which I have made from material obtained from slaughtered animals I have never found bacilli except in a very few cases where the virus was not obtained until after contact with the air, where the vacuum tubes had not been properly sealed, or where the animal was not slaughtered until the last stages of the disease. A photograph of a preparation made from one of these cultivations is reproduced in Plate XI. It seems to be a perfectly pure cultivation of micrococci so far as careful examination with the microscope is able to determine, and it was so virulent that three pigs inoculated with it all contracted the disease and all died.

In my most recent investigations I find that the peritoneal effusion is often impure in the last stages of the disease. In such cases a variety of organisms appear in the cultivations made with this liquid, but pure cultures of micrococci are still obtained from the pleural effusion, or in those rare cases where this too is impure the pericardial fluid and blood have yielded pure cultures of micrococci. A fact of great importance is that no pure cultures of bacilli have been obtained, and that where but a single species of organism has multiplied this has invariably been a micrococcus.

Having obtained such results from my investigations, and having repeated them over and over again, and confirmed them with virus from various parts of the country, I cannot but conclude that swine plague is due to a micrococcus, and that the disease produced by Dr. Klein's cultivated bacilli was a form of septicæmia. And this conclusion is confirmed by the short period of incubation in his cases, and the fact that many of his animals showed no signs of disease other than a slight rise of temperature and an enlargement and congestion of the lymph glands.

The following record of experiments contains the most important of those which have been made since my last report, and is a continuation of the evidence upon which the above statements have been made:

Experiment No. 1.—Two pigs were inoculated June 28, 1883, with virus dried on quills and sent from Indiana. It was obtained by killing a sick pig and immediately dipping the quills in peritoneal and pleural effusion and the exudation liquid from the lungs, and drying this after the manner practiced for preservation of vaccine lymph. In this case the animal from which the virus was obtained did not have a very severe form of the disease. For inoculation the virus on three or four quills was rubbed up with 2cc. of salt solution and injected under the skin of thigh. The fourth day (July 2) there was elevated temperature (102⅘° and 103⅕° F.) and slight redness at the point of inoculation. The fifth day there was diffused redness on the inner side of both thighs, an eruption of small papulæ on the thin parts of the skin and an increased elevation of temperature (103⅖° and 104⅕° F.). July 5 to 9 the temperature remained at or above 105° with one, and reached its highest point on the 7th, being then 105⅗°, and the eruption was very plain and extended over the greater part of the surface of the body. From this time they began to improve, and in neither case was the disease fatal.

This was one of a number of inoculation experiments made to obtain a reliable virus for experimental purposes, and is recorded to illustrate the above remarks in regard to the period of incubation.

Experiment No. 2.—Four hogs were inoculated July 7, with virus also from Indiana, and preserved in the same way as the other, but was obtained from an outbreak which was much more virulent and fatal. This was also suspended in salt solution and injected hypodermically in the dose 2cc. to 3cc. at the inner side of the thigh.

To and including July 17, or for the first ten days, there were small, hard swellings at the point of inoculation, but no positive signs of disease, and the appetite remained good. There were considerable variations in the temperatures, but it is doubtful if this had any pathological significance. July 18, three were evidently sick, with temperatures of 102⅗°, 105⅕, and 106⅗° F.

The one most severely affected was killed July 21, at which time the temperature was 104⅗° F., and there was complete loss of appetite. The point of inoculation was much swollen, the enlargement extending forward under the abdomen, and was about 6 inches in length by 2 in breadth. When cut across it was found to be dense and fibrous and creaked under the knife. A clear lymph flowed from the cut surface. In the center of the swelling was an irregular cavity, 1 to 2 inches across and partly filled with dry caseous material, reminding one of the sequestrum formed in fowl cholera when an inactive virus is injected into the muscles in large quantities. The right lung was nearly all of a deep-red color with extensive areas of infarction. There was a small quantity of effusion in the cavity of the thorax. The intestinal tract was congested but there was no peritoneal effusion.

The pleural effusion was collected in vacuum tubes with all known precautions to prevent access of atmospheric germs, and hermetically sealed. Cultivations were made by infecting sterilized nutritive liquids in the cultivation apparatus with small quantities of this pleural effusion. The cultivation liquids used were pork and beef broths which had not been neutralized and neutral veal broth. All the attempted cultivations were successful, and the organism which multiplied was of identical appearance in each—it was a diplococcus or figure eight in form, and had a tendency to adhere in short chains and small clusters.

This organism was carried through three cultivations, each apparatus containing about half an ounce (15$^{cc.}$) of liquid. August 2 experiment No. 3 was made by inoculating 2 pigs with the third cultivation of this micrococcus. One of these had a hypodermic injection of 4$^{cc.}$ and the other of 10$^{cc.}$ of the cultivation liquid. This was made on the inner side of both thighs and with the latter also between the fore legs.

There was swelling at the point of inoculation within twenty-four hours; but no marked increase of temperature until August 7, when it reached 105⅕° with one, and 104⅘° with the other, with impaired appetite, thirst, and shivering. Two days later the skin over the entire abdomen was wrinkled, flabby, and in places losing its epidermis. From this time they improved in general symptoms until August 17, when the one that received the larger quantity of virus and which had been most severely affected was killed for examination. At this time there was extensive desquamation of the epithelium over the abdomen; the swelling at the point of inoculation had softened and contained pus. There was swelling of the lymphatic glands of the inguinal and mesenteric regions, petechiæ of the serous membranes, and slight peritoneal effusion.

Experiment No. 4.—Three pigs, Nos. 26, 27, and 28, were inoculated June 9 with a cultivation liquid seeded from the virulent effusion of a pig that had died from the result of inoculation with a very fatal virus, received from Illinois. This cultivation liquid contained only micrococci, the appearance of which are very well shown in Plate XI, which was reproduced from a photograph.

June 14, all had elevated temperatures varying from 104° to 105⅖° F., increased thirst, tucked up abdomens, swelling at the points of inoculation, rigors, and secluded themselves in their bedding. The appetite was still fair.

June 20, there was complete loss of appetite, emaciation, and profuse diarrhea.

June 29, No. 27 died, and autopsy revealed congestion of intestines, hepatization of right lung, with abundant effusion in the pleural, pericardial, and peritoneal cavities. Inoculations with this effusion caused death of another pig July 8, after showing the well-known symptoms of swine plague.

July 3, No. 28 was found in a dying condition and was destroyed, in order to get fresh material for examination and for inoculation experiments.

July 6, No. 26 died in convulsions after having presented the characteristic symptoms of the various stages of swine plague.

The notable point in this experiment is the virulence of the cultivated virus. This virus was a pure cultivation of micrococci and produced fatal results in every case. The results of our inoculation experiments with cultivated micrococci have heretofore been more or less unsatisfactory, because, while the symptoms were those of swine plague, the disease produced did not correspond in its malignancy to the swine plague which so frequently decimates the herds of the West. In this case, however, the disease developing as a result of inoculation had all the malignancy of the most severe outbreaks which I have ever witnessed, and in subsequent experiments with virus obtained from these animals this fatal type has been retained and every animal inoculated has succumbed.

On July 3, pig No. 34 was inoculated with mixed pleural and peritoneal effusion obtained from No. 28, which was killed that day in the last stages of swine plague, produced by inoculation with cultivated virus as detailed above. July 15, the temperature was 104° F., and there were periods of shivering. From this time the progress of the attack was rapid; there was a red blush of the skin over the abdomen, diarrhea, loss of appetite, prostration, and tendency to hide in the litter. July 18, it was very much debilitated, the breathing was rapid, and it was scarcely able to walk. It would undoubtedly have died in a few hours. It was killed for examination and for pure virus.

Autopsy showed the lungs to be covered on pleural surface with petechiæ, but there was no hepatization. The inguinal and mesenteric glands were greatly enlarged and congested; the small intestines inflamed; the cæcum was the seat of three large ulcerous patches, 1 to 2 inches in diameter, and several of smaller size. These were black on the surface and on sections the tissue appeared dense, fibrous, and pale. The ileo-cæcal valve was completely covered with such an ulcer, and the mucous membrane of the stomach was much congested.

Thoroughly sterilized vacuum tubes were filled from the jugular vein, from the right ventricle, and with the pericardial and peritoneal effusion, each of which were abundant. At the time of the autopsy small quantities of each of these effusions and of the blood were dried on cover-glasses for examination in the laboratory.

The tubes of peritoneal fluid when opened emitted a very disagreeable odor of putrefaction. Stained cover-glass preparations showed that it contained both micrococci and rods. Cultures of the same contained micrococci, a bacillus with pointed ends, probably the *Bacillus butyricus*, and a few rods of bacterium termo. From this result it becomes an interesting question to learn if these various organisms really existed in the peritoneal liquid at the time of the animal's slaughter, or if they were introduced from the atmosphere during the necessary manipulations for filling and sealing the vacuum tubes. In other words, is it possible for septic bacteria, in diseases which produce lesions of the intestines, to penetrate the walls of these organs and multiply in the peritoneal effusion before the death of the animal? In a former report (Annual Report Department of Agriculture, 1880, p. 432), I have collected a number of observations which seem to answer this question in the affirmative. Fortunately in the case under consideration examinations were made which furnish satisfactory evidence that there were various forms of bacterial organism in the fluid of the peritoneal cavity before the death of the animal. Preparations were made by thoroughly drying this fluid on cover glasses as soon as the abdominal cavity was opened, and in these, of course, there could be no change before examination. Such preparations stained and mounted demonstrate conclusively that while the micrococci predominated, there were also present a considerable number of bacilli. These observations, which were made with the greatest precautions to avoid errors, go far to reconcile the discrepancies which have appeared to exist in the results of the various investigations of this disease.

Cover-glass preparations of blood from the jugular presented no definite bacterial forms even after staining. Cultures of this blood remained perfectly sterile.

The pericardial effusion contained large numbers of micrococci, easily seen both in unstained and stained preparations. Cultivations gave pure growths of micrococci.

The blood from right ventricle showed aggregations of micrococci, and cultures of this blood produced a pure growth of the same organism.

Sections of the cæcal ulcer contained enormous aggregations of micrococci in the depths of the cavity; the necrotic portion appeared to consist almost entirely of these bodies. In some sections small colonies of micrococci were found in the deeper parts of the tissue. No rods could be found even in sections stained with fuchsine, a stain which Klein used in his investigations.

In sections of the spleen, stained in various ways, no organism could be detected.

Sections of the most congested mesenteric gland revealed no organisms within the gland tissue, but the peritoneal surface and its serous covering were studded with micrococci, interspersed with which might be seen a considerable number of large and small rods.

Pig No. 39 was inoculated July 17, and was slaughtered August 11, being at that time very severely affected, and presenting well-marked symptoms of swine plague. The mesenteric vessels were congested, as were those about the ileo-cæcal valve, but there were no ulcerations at this point. The lungs were pale, but contained a number of dark-colored congested patches. There was no pleural effusion; the pericardial cavity contained a considerable amount of liquid, and there was also slight peritoneal effusion.

Cultures of the pericardial fluid gave a pure growth of micrococci. The vacuum tubes, filled with peritoneal fluid, were preserved until September 2, and were then found to contain large numbers of micrococci, but no other organisms.

A large number of observations similar to the above have been made, and in all cases where a pure cultivation has been obtained the organism which multiplied was a micrococcus, and when the virulence of such cultivated micrococci has been tested by inoculation experiments typical and fatal cases of swine plague have resulted.

A NEW FORM OF CULTURE TUBE USED IN THE PRECEDING INVESTIGATION.

The necessity of obtaining absolutely pure cultures of bacteria for the study of their biological properties has led to the development of a very delicate technique, and to the adoption of various forms of culture apparatus by different investigators. To many of these, from the standpoint of to-day, there are almost fatal objections; others, used perhaps with great skill and dexterity by their originators, may fail completely in the hands of those not accustomed to them. To be sure a certain amount of skill and a quick appreciation of possible sources of error must be presumed in the study of the Schizophytes whatever methods and apparatus be employed. The culture tube, described below, which has been devised by me, more especially for the study of pathogenic bacteria, in connection with the contagious diseases of animals, requires, I believe, less of this initial store of manual dexterity and eliminates more sources of error than the great majority of tubes now in use. To those who are engaged in this field of research, the following description and suggestions will speak for themselves.

The culture tube (Plate XIII) consists of a test-tube-like body or reservoir (*a*) of rather heavy glass from 4 or 5 inches in length and $\frac{3}{4}$ of an inch in internal diameter. Over the top of this reservoir a second hollow piece (*b*) is fitted which might be called a cap. Its internal surface is ground to fit snugly over the ground external surface of the upper end of the reservoir, thus forming a ground joint. This cap, 2½ inches long, abruptly contracts near its middle into a narrow tube, having an internal diameter of about ⅜ of an inch. The third piece (*c*) which might be called a ventilating tube is shaped like an inverted U with limbs of unequal length. The shorter limb, about 1½ inches long, fits over the narrow tube of the cap by means of a ground joint. The other, free limb, about 3 inches long, lodges a plug of glass-wool (*d*) from 1½ to 2 inches in length. The tube, itself, when the parts are in position, measure from 7 to 7½ inches. A little sublimated vaseline is rubbed upon one of the joint surfaces to prevent them from tightly adhering to each other after they have been subjected to a high temperature.

The culture liquid is introduced by removing the cap which brings with it the ventilating tube. From 10 to 15 cubic centimeters will be sufficient, though the reservoir will hold conveniently 25 cubic centimeters. The form of the latter may be changed to suit circumstances. The lower end may be enlarged so as to become flask-shaped when micro-organisms, requiring an abundance of oxygen, are to be cultivated. The test-tube form will be found generally applicable, however, by varying the quantity of the culture liquid. Sterilization of the latter is affected in the tubes either by discontinuous boiling or better by raising at once, in a steam-tight boiler to a temperature of 110° C. This method is less troublesome and has been found uniformly successful. The plug of glass-wool should not extend to the free end of the ventilating tube, otherwise it will become thoroughly wet during the process of sterilization.

The pipette, employed to introduce and transfer small portions of cultures, consists of an ordinary glass tube about ¼ inch in diameter and from 2 to 3 inches long, one end of which is drawn out into a very fine, almost capillary prolongation, which must be long enough to reach the bottom of the reservoir when introduced through the narrow portion of the cap. A plug of glass wool (*d*) occupies the tube near the other extremity, which is surmounted by a rubber bulb (*e*).

To transfer a drop of culture liquid, containing the organisms to be sown, from one culture tube to another, the pipette is thoroughly flamed from near the rubber bulb by which it is held between the fingers to the extreme end of the capillary prolongation, until it has been exposed to a temperature of 200° C. and above. It is then suspended with the capillary end down and allowed to cool. Before introducing it into the culture tube the capillary portion is again drawn once or twice through the flame. That portion of the culture tube including the ground joint between cap and ventilating tube is now flamed, the latter gently removed and the narrow portion of the cap flamed. The pipette bulb is compressed according to the amount of liquid desired, the capillary portion introduced through the narrow tube of the cap and some of the liquid drawn up. Before replacing the ventilating tube the narrow tube of the cap is again flamed. The fresh tube is inoculated by introducing the pipette in the same way and allowing a drop to fall into the culture liquid from the end of the pipette, held slightly above the surface of the liquid. The same precautions of flaming the joint and narrow portion of the cap are to be observed. The methods employed in obtaining originally either a pathogenic or other form for cultivation vary according to the habitat of the micro-organism and its association with the other forms, and cannot be detailed here. The method of inoculating the first culture will likewise vary with the nature of the medium in which the particular species is found.

A possible source of error may be looked for in the momentary exposure of the culture liquid to unfiltered air when the ventilating tube is removed for the introduction of the pipette. A glance at the cap (*b*) will convince any one that this source is very small; the tube is quite narrow, making the exposed area a minimum. Moreover, investigators in general agree that brief exposure to the air of a room where dust is kept well under control is of no practical consequence. The chief danger lies in the use of instruments not thoroughly sterilized. Another possible source of contamination may be looked for in the unfiltered air drawn into the pipette as it slowly cools and the contained air contracts. During the flaming the contained air will usually expand to nearly twice its bulk. Hence 1 cubic centimeter of unfiltered air will be drawn into a pipette of 2 cubic centimeters capacity. This danger is practically absent when we consider the following: 1. The dependent position of the pipette during cooling opposes the force of gravity to particles of dust. 2. Most of the unfiltered air is expelled and replaced by filtered air when the bulb is compressed for the purpose of drawing up some of the culture liquid. 3. A sufficient amount is to be drawn into the pipette so that both the first and the last drop need not be used for sowing, but only that portion of the liquid which has not been in contact with the air. 4. If we calculate the chances of contamination from 1 cubic centimeter of unfiltered air, aside from all the favorable influences just mentioned, by allowing 1,000 germs to the cubic meter of air, which is a very high estimate, we shall find them to be 1 in 1,000. The introduction of unfiltered air may be entirely prevented by sterilizing in another way. The bulb is removed, the capillary extremity sealed, and the entire pipette thoroughly flamed as above while it is held with tongs or forceps. The air will then be filtered as the pipette cools, since it can enter only through the opening which is guarded by the plug of glass-wool. The rubber bulb is replaced in such a manner as to cause a slight increase of pressure within the pipette. On crushing the sealed extremity with flamed forceps there will be a slight outflow of air. Prepared in this way the pipette will contain none but sterilized and filtered air. A number can be kept sealed and ready for use if desirable. Another device to accomplish the same end is the following: A cylindrical glass jar is closed with a thin rubber cork which is perforated by a number of holes large enough to lodge the wider part of the pipette. These holes are kept closed with glass rods or tubes plugged with glass-wool. The air within is supposed to be free from dust. After flaming the pipette it is introduced into the jar beyond the capillary portion, by removing one of the plugs, and there allowed to cool. In this way the pipette will become filled with air practically free from dust. Though we have used the culture tube continually for nearly a year without resorting to these devices except for special purposes, we do not recall a single impure culture derived from a preceding pure one. Contrast with this the statement made in 1881, by one of England's most experienced experimenters in the field of pathogenic and septic bacteria: "So in the room in which I work I have never been able, without the aid of the spray, to transfer micrococci from one flask to another, for in the latter flask *bacteria* almost invariably developed.*

The difficulty experienced by Dr. Cheyne can only rest with the inefficiency of the culture apparatus employed.

The culture tube recommends itself also for the readiness with which it is filled, sterilized and inoculated. A perusal of the experiments of elder investigations is sufficient to impress this upon the mind. No plug need be disturbed, no tubes sealed or broken, no spray or antiseptic dressings employed. Its manipulation is safe, as no sharp jagged edges need be feared when a dangerous culture is being prepared. The tubes are readily cleaned, and may be employed over and over again, so that the initial cost cannot be cited as an argument against them, and in favor of homemade apparatus, which requires outlay for material and time for its preparation. The form of the reservoir is very well adapted for a careful macroscopic examination of the culture. The faintest turbidity, or cloudiness is readily recognized. The concave bottom reveals the earliest trace of sediment, while the beginnings of a surface membrane are as easily detected. Perhaps the greatest advantage consists in the facility with which small portions may be withdrawn, more especially for repeated microscopic examination. Those who have devoted much time to the schizophytes will certainly appreciate this. By proper management of the pipette, portions of the deposit of the liquid or of the membrane, if this be present, may be withdrawn separately for examination. This differentiation, as it were, of the culture is greatly favored by the form of the reservoir. Portions of the membrane are more conveniently withdrawn with a looped platinum wire or a needle.

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*Antiseptic Surgery, p. 261.

Another use to which the tube may be put will suggest itself by an examination of Plate VIII of the *Mittheilungen a. d. k. Gesundheitsamt, Bd. II.* Koch employs for his cultures on solid substrata a simple test tube, the mouth of which is closed by a plug of cotton wool. The culture tube described above can be used for solid media equally as well, if we discard the ventilating tube entirely and introduce a plug of glass wool into the narrow tube of the cap. When the gelatine or blood serum is to be inoculated the cap is removed and held so as to become an efficient screen from floating dust during the operation. Moreover, the gradual desiccation of the culture medium inevitable in the simple test tube of Koch may be reduced to a minimum by covering the narrow portion of the cap with a rubber bulb, such as caps the pipette (*e*). Finally, if any doubt should arise as to the imperviousness of the ground joints to aerial germs, it might be said that of the hundreds of tubes containing sterilised fluids of various kinds and stored away for many months, none have shown signs of a spontaneous infection.

Description of the figure (reduced one-half): I. The culture tube with the parts in position. II. Parts of the culture tube separated; the extent of the joint surfaces is indicated by dotted lines. III. The pipette; *a*, reservoir; *b*, cap; *c*, ventilating tube; *d*, plug of glass-wool; *e*, rubber bulb.

GREAT PREVALENCE OF SWINE PLAGUE.

Swine plague has prevailed to a wide extent during the past six months, and the losses have been enormous. The investigations of this disease are being continued, but the great demands made upon our time in the investigations of cattle diseases, previously referred to, have prevented us from reaching any practical results, and have not allowed us to properly test the many proposed remedies which have been brought to our attention.

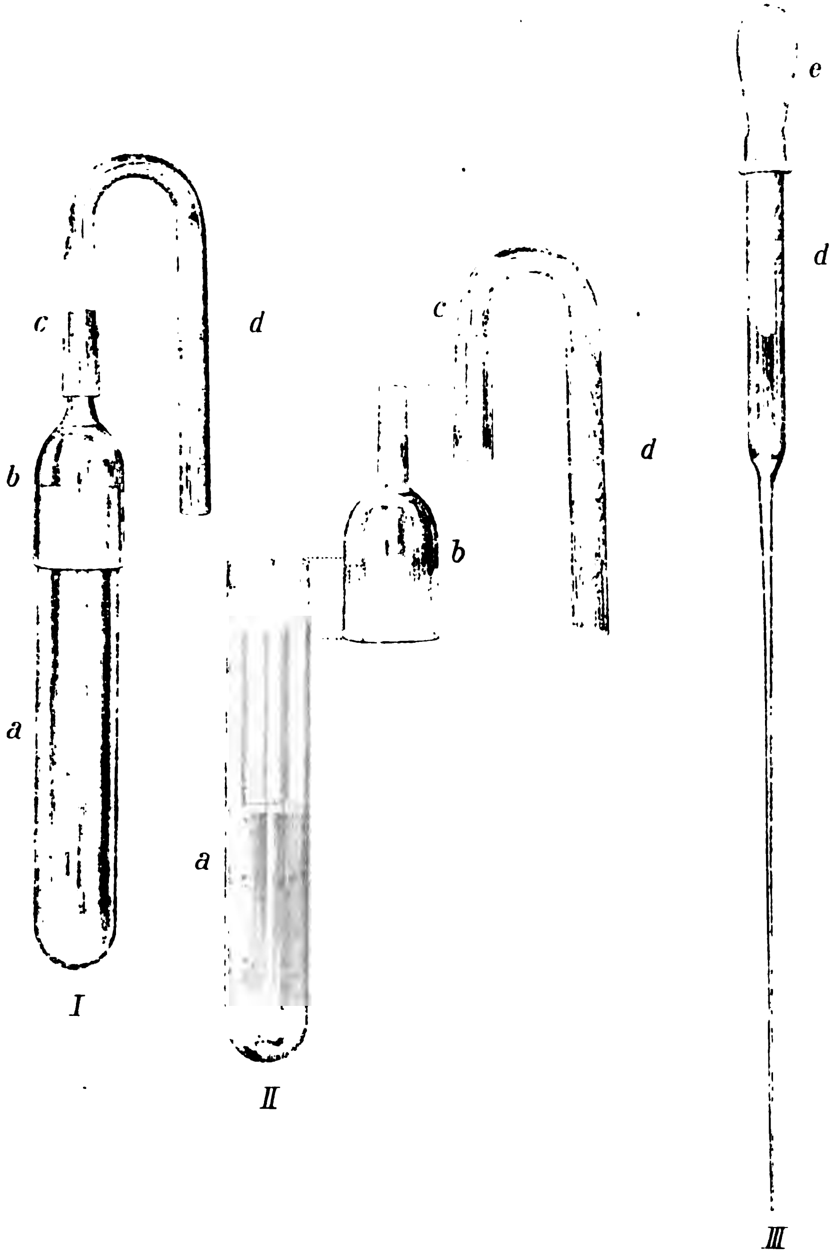
Respectfully submitted.

D. E. SALMON, *D. V. M.*,
Chief of Bureau of Animal Industry.

HON. GEORGE B. LORING,
Commissioner of Agriculture.

NEW CULTURE TUBE.

PLATE XIII.



CATTLE INTERESTS WEST OF THE MISSISSIPPI RIVER.

REPORT OF HON. J. B. GRINNELL, OF IOWA, AGENT BUREAU OF ANIMAL INDUSTRY.

Hon. GEORGE B. LORING,
Commissioner of Agriculture.

SIR: I have the honor to present herewith a report, by your direction, under the requirements of the law enacted by the present Congress creating a National Bureau of Animal Industry.

The specific field assigned for my investigation was our country west of the Mississippi River, embracing twenty-two States and Territories, and an area of 2,203,000 square miles, equal to 1,409,000,000 acres. I have given the most of my time for the last six months in personal observation and study, in order to answer specific questions presented by your Department, and have discussed these cognate questions relative to the cattle and their home west of the Father of Waters. I can, owing to a lack of time, make only partial and incomplete statements relative to the extreme Northwest and the South, yet indulge the hope that this paper may be of value to the inexperienced, a guide to those seeking investments in animals and ranches, and furnish a just occasion for the citizen to guard and improve a national heritage to be valued, not more for its grand and imperial area than for the latent sources of wealth found in varying climes and of magnificent adaptations.

SOURCES OF INFORMATION.

I have drawn on my thirty years' experience as a resident in the Northwest occupied with cattle husbandry, and in association with some of our eminent stock-growers. Observations have been made on long journeys, in carriages and by railway the present year, equal to 14,000 miles; and trips previously equal to 7,000 miles through the ranch country, were the occasion of a work, "The Cattle Industries of the United States," by the writer hereof (now out of print) which I have drawn from as original matter. Surveyor-generals and land officers have aided me in forming a reliable estimate of the capacity of our domain, which has been supplemented and confirmed by answers to hundreds of circular inquiries, addressed to intelligent frontiersmen and successful breeders, whose opinions may be regarded as the highest authority on the subjects herein discussed.

THREE NATURAL TERRITORIAL DISTRICTS.

There are west of the great river three grand divisions of territory formed by adaptations to certain grains and grasses, which have led to the adoption of various methods in our cattle industry. The first division is the corn belt, embracing the States of Iowa, Missouri, and also a portion of the river bottoms of Arkansas, Louisiana, the Indian Territory, and the eastern half of Kansas and Nebraska and the southern half of Minnesota, approximating 250,000 square miles. In this district there are eight millions of cattle, the care of which constitutes the chief industry and source of wealth of six millions of people. This interest is yearly intensified, and becomes more apparent in judicious breeding, increased shelters, better food, in the choice of a variety of grasses, and a resort to ground or steamed grain for economical beef production. There are local and State stock organizations, formed for mutual benefit in exchange of opinions, and in the publication of volumes rich in the experience of gentlemen who have aspired to eminence as breeders of improved stock, while finding relaxation and rest from the exacting labors of professional life and public service, also a fair return for care bestowed and capital invested. The animal has been improved and the avocation of his owner elevated quite to the dignity of a profession becoming at once honorable and attractive to the well-educated and ever aspiring youth now successfully contending for the prizes so long borne away in the older States.

A second division would constitute the grazing district where tame grasses are not in general use, and the native herbage is made into hay and utilized with straw for winter food without grain, there still being large tracts of land, as in Iowa, Missouri, and Dakota, adapted to corn, but as yet untilled. This area, including portions of several other new States, is illustrated by the multitude of hay-stacks and rude sheds extending over a country equal to 150,000 square miles, now supporting 3,000,000 of cattle, large numbers of which are annually driven or freighted eastward to be ripened on corn. Under these conditions there is a large individual ownership, but a longer time required for winter-feeding, owing to the early destruction of prairie grasses by frost and a later spring growth than the domestic grasses. The enterprising owners are each year, by sod-turning or close feeding in inclosures, improving their pastures and infusing new blood in their herds, which makes a fair exhibit on the balance sheet when stockers find ready purchasers at $3 a hundred and upwards.

The third grand division is the range country. This comprises arid and desert plains, one of which I crossed for 100 miles, remarkable only for a crop of sage brush and the absence of a drop of water; another a distance of 150 miles, where not a blade of grass was visible, nor insect, bird, or creeping thing, nor water in stream or pool, neither of which wastes will I locate, but not to be overlooked in a just description of a country relieved of this view of desolation by fine tablelands, rich ravines, sheltered by overhanging rocks, mountain gorges, and parks, fruitful of evergreens, forests, adjacent to foot-hill springs and flowing streams, lined with willows, a refuge for the herds in time of storms.

The State of California must be excluded from the range country proper, safe and profitable as cattle raising has been in some sections. Fruit growing, wine making, sheep husbandry, and wheat raising have denied expansion on the Pacific coast to such an extent that California looks to the border State of Nevada rather than to its own ranches for a supply equal to its local demand.

I submit the following table, with names of States and Territories, and the acreage of each, the number of cattle, the acres occupied by each, and their total cash value, waiving the mention of fractions, since round numbers will make an impression as to the vastness of our domain and the room for legitimate bovine expansion:

States and Territories.	Acreage.	Number.	Acres occupied by an animal.	Value.
Texas	175,000,000	5,500,000	32	$92,000,000
Oregon	61,000,000	601,000	101	16,000,000
Nevada	66,000,000	234,000	300	6,000,000
Colorado	67,000,000	815,000	82	22,000,000
Arizona	73,000,000	216,000	340	4,500,000
Dakota	95,000,000	346,000	270	7,220,000
Idaho	54,000,000	220,000	247	5,900,000
Montana	93,000,000	687,000	136	18,100,000
New Mexico	78,000,000	700,000	110	14,400,000
Utah	54,000,000	170,000	318	4,400,000
Washington	44,000,000	270,000	160	7,800,000
Indian Territory	41,000,000	520,000	80	8,840,000
Wyoming	60,000,000	900,000	66	23,600,000
Total	961,000,000	11,179,000	87	230,260,000

In the States and Territories above named there are found between 8,000,000 and 9,000,000 of cattle guarded and owned by a people illustrating the extreme habits of our national life and character, but no more divergent than the theories and practices where a favorite industry finds its greatest enlargement and meets with most apparent success.

I have named 8,000,000 as the number found in the corn belt and first division, and 3,000,000 in the intermediate native-grass section, and 9,000,000 for the ranges, exclusive of California, an aggregate of 21,000,000, and total cash value of $540,000,000. The range estimate must be, in a measure, conjectural, since the assessor cannot count and inspect the herds; besides, the agents and owners, in view of taxation, will claim the benefit of every doubt when asked for the total ownership. From original sources I find less than 7,000,000 on the tax lists, and the best local authorities do not deem the adding of 2,000,000 to the estimated lists an overstatement as to the numbers, equal to a grand total of 21,000,000 cattle west of the Mississippi.

SPECIFIC INQUIRIES.

These I have made under your direction, not in the belief that my conclusions will find universal indorsement, but they should be credited as pioneer efforts in a new and specific field of research, where there has been a candid inquiry without personal interest or prepossession as to breeds, methods, or localities.

1. Acres adapted to pasturage west of the Mississippi river.

Official reports of surveyors and the opinions of herdsmen as to their localities, after deducting the corn section, desert wastes, and inaccessible mountains, vary from 500,000,000 to 1,000,000,000 of acres, with a preponderance of testimony at 800,000,000 of acres.

2. The number of cattle they would sustain.

If an allowance is made for the present grain crops and the usual complement of other animals incident to present farming methods, there would be one answer—to give the full capacity there would be another. The corn and native grass districts, after an allowance for grain and other animals, besides neat cattle, and including wastes and forest tracts, should support one bovine for every 8 acres, or 22,500,000 head, near double the estimate for 1884 in numbers.

The range country presents, as a whole, an interesting field of inquiry as to the number of acres required for one bovine. Enthusiasts who take a rose-colored view of the growth and capacity of the soil which bounds their abode name 20 acres as a fair average. Another class of minds, deprecating overstocking as perilous in seasons of drought or snow-storms, when but a limited surface may be left bare, make an estimate of 50 acres. The experienced pioneer, in contradistinction to the "tender-foot," holds that close grazing is destructive to buffalo and bunch grass, and that noxious weeds and sage-brush will cover the surface, save on the limited moist soils, and that 80 or 100 acres is a safer allowance. Striking an average of 40 acres to an animal will give the range country a capacity for the maintenance of 34,000,000 of cattle, or about four times the present estimated number of ranch stock, and more than a justification of foreign opinions that our ample pasturage is equal to a supply of the beef-eating peoples. The London Times gives this assurance:

> For the additional millions the North American continent is destined to support, there are commensurate millions of acres waiting to yield meat for cattle and meat for man.

3. THE NUMBER DEVOTED TO DAIRY PURPOSES.

There are 3,000,000 of animals used for dairy purposes, the State of Iowa alone having more than 1,000,000 of cows, leading any State in the Union save the State of New York. Of later years, the high price of young stock and a desire for an early growth has allowed a larger number of calves than formerly to follow their dams for the season. This custom, which is not confined to the South and Southwest, has lessened relatively the number kept exclusively for dairy use.

4. THE MOST ACCEPTABLE BREEDS.

Near towns and cities the Jersey, Alderney, and Guernsey are favorites, and in the cheese-making districts the Holsteins have a good repute. Mixed bloods of the above and certain families of the Short-horns give eminent satisfaction to the dairymen.

Spanish stock, called Texan, with high-blood crosses, predominates Southwest, while northward there is a large percentage of animals that show desired and valuable points for milk and stock not common to the native. With the great increase of our dairy products there is a close selection of the heifers giving promise as milkers, and inferior ones, furnishing beef preferable to that of the grass-fed steers, are sent to the local butchers. Aside from all fancies as to name and form, quality and quantity of milk establish the favorites.

5. THE FAVORITE BREEDS FOR BEEF.

They will stand in this order and as to numbers: Short-horns, Herefords, Polled Angus, and Devons; but not as to the enthusiastic commendation of their owners, since the Herefords, Angus, and Devons are leading on the ranges for the desired "rustling" qualities, and giving value to the hardy characteristics in a "survival of the fittest" after severe droughts and long winter storms. While it is my province to give opinions, rather than to criticise their authors, this explanation may be pertinent to the question. All the animal kingdom is subject to the laws of heredity. None in their growth and development will escape the effects of climate, care, domestication, and training. Nature provides a coat for the fur-bearing animals best suited to their necessities, under the same law that supplies the buffalo, of the bovine race, with the dense long hair protection necessary to his existence in northern climes; besides, certain exposures contribute to hardihood, vigorous reproduction, and tenacity of life. On this postulate it is said the best Short-horn families fare sumptuously; their limbs are little used in locomotion, and they are bred for and attain a fine, delicate coat of hair, which for many generations will be transmitted under law, whether enjoying princely care, or exposed to storms, scant fare, and trials of acclimation on the cattle ranges. If it is good care and judicious breeding that has achieved for the Short-horn a world-wide fame, it does not follow that a radical change in food, climate, and neglect will continue them especial favorites under a new *régime* and ownership where fancy is overlooked for prosaic utility involved in numbers branded and a net return of dollars.

The advocates of the other breeds, new candidates for favor, assert that they are built for endurance, and sprang from unpampered races that knew little of warm stables and rich herbage, inheriting habits and a coat of hair which give them organic adaptation and great value for hardihood, prepotency, and those qualities which are of vital concern to the ranchman, who waives simple theories from notice in the substitution of facts.

6. AT WHAT AGE ARE CATTLE TURNED TO MARKET?

The period is determined by locality and the varying methods of husbandry and breeding. In Texas, Oregon, Washington Territory, and on the plains an interest account, the trivial cost for care and lessened peril to the well-grown steer, are important factors. Five years is not an early age, nor six and seven a late one, to market range beeves. In those districts where summer herding and prairie-hay feeding in winter are practiced, the age of four years is the favorite age for marketing. This is based on the cost of $1 a head for summer care, and the need of two tons of native hay for the winter, at a cost of $4. This estimate is made at the latitude of 41° and northward, and insures good profits where there is a small outlay for buildings and a trivial interest item for land, and autumn sales of grass-fed stock are made at from $2.50 to $3 a hundred on foot.

The time varies in those sections where cultivated grasses and corn are the staples of food. Here there is a growing opinion and a decided conviction that early maturity and shipment, say at two or two and one-half years, is the best policy. High bloods reach, with good care, 800 pounds live weight at 12 months, 1,400 pounds at two years, and from 1,600 to 1,800 weight at two and one-half years. The next six months or year following this period, there is less flesh taken on relatively than before, while an increased amount of food is required, and a contingent item in an interest account which further stimulates early maturity and marketing.

7. WHAT IS THE ANNUAL INCREASE OF CATTLE?

Eminent authorities in the various States and Territories differ as to the per cent. Increased values have led to an early marketing of cattle of both sexes. A movement of young heifers from Minnesota, Missouri, and Iowa to the Western plains, notably to Montana, where not less than 100,000 during the year 1884 have been shipped over the Northern Pacific Railway, has lowered the ratio of increase in the older States. Louisiana, California, Nevada, Oregon, and Utah show but a small per cent. of increase, if any, taken as a group. The drought in Western Texas, and severe storms and insufficiency of water northward, taken with the well-known losses by acclimation, will lower the per cent. of gain given in news paragraphs and by enthusiasts. The talk of a "calf crop" of from 60 to 75 per cent. must be largely discounted, for it is not forgotten that the unseasonable birth of tens of thousands of calves dropped in snow or at a temperature below zero, destruction of the young by wolves, and a fearful loss of emaciated, overtaxed young mothers, form an aggregation of losses which will only cease under better methods hereafter to be noticed. Great as they are, accounted blunders and neglect combined, the rise of stock in value, and the occupation of virgin tracts, well sheltered, have brought fabulous profits to the skilled and enterprising, even on the small annual increase of 8 to 10 per cent., which I name as the highest justified by the yearly increased shipment of beeves, and an appeal to the last decade and census, with a belief that the enumeration of 1890 will not give us above 30,000,000 head west of the Mississippi, and 54,000,000 in the Union. This would become an increase of 18,000,000 head the present decade, against 12,000,000 in the past.

8. WHAT IS THE COST OF SHIPMENT PER CAR TO KANSAS CITY, TO SAINT LOUIS, OR CHICAGO?

As as rule the greater the distance from the great stock marts the less the cost per ton by the mile or by the car-load. A load is from 17 to 20 beeves, estimated at 20,000 pounds. Three hundred miles west from Chicago or Saint Louis the freight rate is from $50 to $60 a car. Three times the distance, *e. g.*, from Denver, Colo., or Cheyenne, Wyo., the rate is from $120 to $130 the car. A lower rate obtains relatively for a longer distance in the Southwest and on the Northern Pacific Railway, while a fair average would be a cost of 8 mills per ton per mile, or $3 a head 300 miles distant from the principal Western stock-yards, or 4 mills per ton per mile for 1,000 miles. A lighter average weight of beeves from the ranges would entail for car lots $7 a head for transportation. A longer haul, in railway phrase, often legitimately shows a relatively less per cent. in cost.

9. A COGNATE QUESTION RELATED TO PROFITS IS THE COST OF REARING.

In the best corn districts it is estimated that well-formed, grain-fattened steers cost $5 a hundred, live weight. Those grown on grass and hay alone at $3 a hundred. Range cattle, it is estimated, cost from $1.50 to $2 a hundred, determined in a measure by the value of parent stock and according to the varying fortunes incident to fair or inclement seasons.

10. THE CONDITION OF CATTLE THIS AUTUMN, THE MODES OF SHELTER—AND GAIN OR LOSS OF ANIMALS DURING WINTER—AND THE PERCENTAGE OF LOSS FOR WANT OF SHELTER.

Not for many years have the cattle been in as good condition for wintering as the present. This is owing to exemption from cold storms, and a heavy rainfall which hinders the curing of grasses, which are the reliance for winter food.

As to the modes of shelter in the older States, an open rail fence is no longer deemed an economical or humane shield from storms, and poles covered with straw or coarse grasses afford comfortable sheds which better taste and a heavier purse supplant with commodious barns. In the newer sections there is still, in disregard of all economic considerations, a great neglect, and on the ranges sheds as a rule are an exception save for young stock and emaciated heifers which may receive care in the perilous spring storms.

The question of gain or loss during our winters does not allow a divided opinion. In the older States, excepting housed stock, there is a loss of six months feeding, animals weighing less the last of April than on the last of the preceding October, and this is not a full recital of the loss, since a low condition forbids, for weeks, recuperation and thrift on luxuriant pasturage. In the newer States it will be claimed that there are exceptions to this rule. They are found in the South, and on the Pacific coast, where the cattle are few, and natural shelters, springs, and cured grasses are abundant. In the Northwest epicurean taste finds an exceptional change from juiceless "blue beef," where there are warm Pacific currents known as the "Chinook" or Japanese winds, which remove the snow in a few hours, and unlock the streams from a long, icy embrace. This is a common phenomenon in late winter, and quite the rule in early spring where the altitude is favorable to warmth, and multitudes of cattle find food and comfort in natural shelters hardly surpassed by the improvised shed or capacious barn. Under these conditions there are gains in the winter rather than a loss in weight.

I forbear to awaken local discussion by a designation of the sections where the greatest losses are sustained for want of shelters. In the older States it cannot be that on an average less than 5 per cent. of the calves and weak cattle are missed in the spring count, while cattle in the native-grass sections, because kept longer on dry food, show even a greater mortality. On the open ranges shelters are deemed impracticable, and before storms cattle drift often a hundred miles, hungry and thirsty, to a broad river covered with glare ice, which they cannot cross, and there huddle and perish. A want of water, in the opinion of Mr. Swan, of Wyoming, one of the eminent authorities, causes the death of ten cattle for every one dying because deprived of food and shelter. Many owners claim no greater loss than 3 per cent. on the plains, and others admit a decimation of the herd equal to 25 per cent., which a water-supply and temporary shelters would have in part averted.

There is less exposure and loss in the broken forest sections where the high mountain ranges forbid, as a rule, heavy snowfalls on the slopes eastward, and from 1 to 3 per cent. is the estimated loss. Striking an average will give not less than 5 per cent. as the annual loss for want of shelters, which the forecast and labors of ranchmen might avert.

11. IF ANIMALS HAVE DISEASES, WHAT ARE THE LOSSES BY THEM AND METHODS OF PREVENTION?

There are no diseases west of the Mississippi not common to most stock-growing countries, and none of these are contagious, excepting what is known as splenetic or Texas fever. It is denied that this is dreaded as a local disease, and is only to be feared when the Spanish stock are driven from their native home and have left their *excrementa* to be brought in contact with northern cattle. Contagion and death, it is believed, will be averted if the two classes of stock are only brought into close quarters after the ground on which they rest is frozen. The protestants will not forget that the movement of cattle in railway cars has been the occasion of severe losses in several States and Territories during the shipment of 1884, aggregating thousands, inciting a turning back of numerous droves from the North by threats and violence, besides calling forth proclamations which give effect to State and Territorial laws, forbidding the ingress of Spanish stock until after frosts. Well-attested cases of disease and mortality awakened fears that the infected might clandestinely be brought to the shambles, and for a time there was a reduced consumption of beef by 10 per cent. in the city of Denver and other localities, and as a further consequence a lull in cattle and ranch sales, also forecasting a dreary outlook for an early and profitable expansion.

A national trail, allowing at all seasons a drive northward from the breeding districts south, as a device to prevent the spread of disease, has zealous advocates. It must encounter serious oppositions in securing favorable State and national legislation, in fixing its terminus, removing local distrust, and in causing serious obstruction to local driving east and west, besides a burden in the sum of millions of dollars in the line of condemnation and appropriation of a tract of land equal to four to ten millions of acres, of course to be fenced or held in rigorous quarantine.

The railway scheme as a means of conveyance would seem to be the more practical but for fear that the Canadian railway might attract business to its line, and in addition encounter the jealousy of rival companies whose profits might be lessened. Then, could the necessary capital be enlisted? The gross business must be conjectural, in view of the increasing capital at the ports on the Gulf of Mexico and New Orleans to be used in facilitating beef and stock exportation. The railway lines in the frontier States are not now remunerative, and will another be added to the list?

Winter driving of steers will be found a practical solution of this question. It avoids, at the outset, cost and the necessity of corporate action and State legislation, the legality of which could not be determined, on appeal, for years. Nature in topography, by the shelter found east of a chain of mountains, and the supply of water insured in seven large streams and numerous tributaries to be crossed, has aided in this inquiry. From Red River, in Texas, to the Powder or Big Horn River, in Wyoming, it is but 600 miles, and the streams to be utilized in the way are the Canadian, Cimarron, Arkansas, Smoky Hill, Republican, North and South Platte, and other water supplies to be reached by wind-mills and artesian wells. Hay, where most needed in the northern section, is free and abundant on the low lands. Five east and west lines of railroad would be crossed and readily utilized in bringing food where there was a failure to find grass on the way. The alfalfa, a plant recently cultivated, which produces by irrigation several crops in a season, and from three to five tons to the acre of nutritious hay, could be grown as a cheap forage and in abundance to meet the wants of the well-grown steers of an age to escape the perils of acclimation. This plan, as a whole, could be easily and cheaply tested.

12. THE SHIPMENT OF DRESSED BEEF.

This question, in its varied aspects, is one of practical interest. As a new enterprise it has enlisted several millions of capital. I found in my investigations a well-known gentleman, Marquis de Mores, at Saint Paul, at the head of a company, which is extending its operations from that city a thousand miles to the Yellowstone River, having at favored localities cattle-yards, ice-houses, and cooling rooms, from which cars are loaded with beef of the best quality, kept at a fixed low temperature, marketed in advance for the eastern cities, and to be delivered on a given day, a portion finding its way in good order by steamer from New York to the French and English markets.

In Chicago the daily slaughter, by different firms, for refrigerator beef aggregates 2,600 head, with an increasing popular demand which has at an early day awakened the cry of "dressed beef monopoly." The charge, it would seem, cannot long in reason be made, since there is no patent on the process. Ice is free, cattle can be found in the open markets, and there are numerous railways seeking business as common carriers to meet the wants of many remote cities.

These are the points made against the prevalent live shipment. Animals become fevered by thirst, hunger, and weariness, and bruised by the changes and perils of a long journey. The shrinkage is from 5 to 7 per cent. against 2 per cent. on beef quarters in transit. Cost of attendance, food, and yardage is equal to from $1.50 to $2 on a bullock, all saved in refrigerator shipments. The freighting of live stock is more expensive than that of beef quarters, owing to penalties incurred, often for delays, and the lack of standing room to allow cars their maximum tonnage. Gravest of all objections is the unnecessary cost, in a gross shipment, of from 40 to 50 per cent. of the animal in offal to the seaboard, while a large fraction of this amount has equal value west to the manufacturer, and for swine-feeding on the frontier. In favor of the new process it is held that the time required for beef shipment assures the needed interval between the slaughter and consumption, besides the normal temperature and healthy condition at the ranges is not possible under the changes and emaciation incident to a journey of 2,000 miles. There follows in deduction cheaper food for the consumer, which increases its consumption, and better prices in a fair field of competition for the producer by from $4 to $6 a head on average beeves.

What is in the way of the desired development? The interests of long-established butchers and commission men, a systematic and untruthful decrial of the quality of refrigerator beef on the slightest occasion, and the opposition of the owners of stock-yards. A greater discouragement is found in the arbitrary rates fixed by freighting and railway companies, seemingly oblivious of the rights of parties who would take advantage of a modern device and experiment. As an example, I give the latest official decision in an adjustment of rates on dressed beef and live-stock shipments from Chicago to New York. It is on the basis of 32 beeves, weighing 40,000 pounds and costing $2,000. Proper credits and charges are given and made against the two classes of shippers, and when the sales in New York are computed dressed beef is made to pay in freight a difference of $156.98, equal to a fraction less than $5 a head on the 32 animals.

Writing in the interest of cattle husbandry, I deem it my duty to say that the above adjustment is in the line of an assumption that flour should pay the same freight as wheat unground; oil from flax-seed the same as the seed before crushed and separated from the cake, and the corn crop, shelled, to have no advantage in freighting over a greater bulk with the cob and 20 per cent. added weight. In the case of beef, there is greater apparent injustice in the policy and demand that the charges on the shipment of lives tock shall be maintained in application to a less weight and real cost, in disregard of invention and enterprise which should inure to the benefit of consumer and producer equal to $5 a head, or the sum of $1,000,000 annually. The magnitude of this yet infant industry I have given, and the benefits to accrue to the masses are so apparent as to require no further mention.

13. FACTS AND OPINIONS RELATIVE TO THE FUTURE OF OUR CATTLE INDUSTRY.

The rearing of 9,000,000 of range cattle has been attended with obstructions and losses. Railways have in all directions aided in overcoming distances, added to the value of mature stock, and there are twenty years of rich and varied experience behind the ranchmen. Pains and gains, as in all material relations, have been found in close alliance; witnessed in the infusion of new blood, which has arrested the deterioration of native stock; fixing the location of the more valued muscle (meat) and increasing the weight of beeves. There is a steady migration to the districts abounding in ravines, "cooleys," timber, springs, and mountain passes, to escape from our stocked and smooth grazing unprotected areas, the potent causes in the decimation of herds. A larger number of skilled riders will be required, and an increased cost in the "round-ups" will accrue; thieves, whites and Indians, by unlawful branding and stampeding will, with less peril, ply their arts; owners of small herds by springs, hitherto in fortunate seclusion, without enterprise or capital to furnish their quota of herding stock, will negative plans of improvement and be overborne under border law, which denies a larger area than necessary for actual use; many will sell under pressure; others will resort to a wanton destruction of property and a burning of the richest grass districts. Tests of endurance by even the larger owners will be sharp and multiplied; encroachments by strangers will become a bar to the comity and fraternity of civilized life; fences built in good faith will be despoiled. Amidst the constant fears and expectations of removal the breeding bulls, restrained by compact during the months of July, August, September, and October, will be at large, forbidding a profitable increase. The losses by overstocking, and the strifes engendered by a system of "all things in common," refers us to the early oriental cattle-kings, but does not find a remedy for our evils.

"Abraham said to Lot, Let there be no strife between me and thee and my herdsmen and thy herdsmen. Then Lot chose all the plain of Jordan, well watered everywhere; and separating, Abraham dwelt in the land of Canaan." They were brethren; the country was rich in springs and grasses, and without adventurous rivals, while our domain is scant of water, and great numbers are seeking the best, stimulating strife and violence, a bar to permanent location, and success will therefore be found attainable only under a new policy.

THE GOVERNMENT SHOULD LEASE TO CATTLE-RAISERS THE LANDS NOT ADAPTED TO AGRICULTURE.

I am aware that it will be said that this policy strikes a fatal blow to the pre-emption and homestead laws, and would favor monopoly. A complete answer is: There are hundreds of millions of acres in other sections awaiting occupation by the landless, and the few settlers are now overborne by the large foreign and American land and cattle companies which have not the spirit of monopolists, but are encouraged to occupy without limit or cost, when able to be of service to the pioneer of limited means, and anxious to lease for a fixed cash rental, ranges now occupied or those without a claimant.

All the details under an enabling act of Congress as to the limits or renewal of leases, the areas determined by present occupation or natural divisions, could be left to commissioners from the respective States and Territories, who would not fail to gain the hearty co-operation of the corporation controlling not less than 100,000,000 of acres of land, not agricultural tracts or included in railway grants. The domain adapted to a trial of this policy would include the States of Colorado and Nevada and the Territories of Arizona, New Mexico, Utah, Wyoming, Idaho, and Montana, equal to 472,000,000 acres. Allowing 72,000,000 for present occupation and to be controlled by purchase, there would remain 400,000,000 acres through which course many large rivers, ample in their flow, and by the use of wind-mills, artesian wells,* and irrigation, to cause grasses and grains to appear where now there is only an occasional blade of nutrition or entire barrenness.

The Arab with French capital has become our instructor, causing water to be raised far out on the desert of Sahara, realizing more than a poetical oasis—a thousand gardens cultivated by happy dwellers in towns shadowed by groves of palm trees; while in our own country, under more favorable conditions, the city of Greeley, Colo., and many others have risen by the aid of water on desert plains. The fine growth of grass and field crops north of, and in the region of, Salt Lake, also the great canal of Southern Arizona, alike indicate what private enterprise and combined capital will do under a leasing system in reclaiming sterile sections brought in contact with water. That species of clover known as alfalfa, nutritious and of wonderful growth, will become by the use of water on the leased alluvial tracts a staple food of inestimable value for the emergencies of winter. I but mention further benefits to follow the leasing policy, better breeding of stock; increased fencing, where the trees have grown suited to wire; by millions of acres in the interest of labor and economy in herding; an increased capacity by a better growth of food and full occupation equal to fourfold 16,000,000 of cattle, where now there are only 4,000,000. Stability and confidence, which would insure better buildings, greater care of stock and less risk, and a tax of from 1 to 5 cents annually per acre would be no burden, yet, aggregating from $5,000,000 to $10,000,000, ample to meet all the expenses of local and State government and a surplus equal to the demands of schools, universities, and humane institutions. The gain in the certain advance of society is a great concern. Home life in the cluster of towns and villages by the streams and marts of trade would, in a measure, supercede roving, unsettled, nomadic habits, unify the people, and give such stability to society as is found in Australia under the wise policy of leasing grazing lands adopted by Great Britain. The Secretary of the Interior advises a policy of leasing Indian reservations not required for occupation for the benefit of the nation's wards. Will not our National Congress adopt the same policy in order to promote peaceful and desired occupation, and to weaken the might of monopoly, by an equitable apportionment of the ranges, that in the future it may be of more value to the occupants and the nation than it has been in the past?

Cattle and ranch companies, as constituted in the western country, have become large and powerful organizations, promotive of comity, safety, and real progress. Since misjudged in the distance, it is due to their stockholders and officers that I should give them credit for a pacific and liberal policy that they do not oppose, but favor the policy of leasing the lands to avoid strife, and promote better society, securing equal gains for stockmen in the end, while creating a cash revenue. My duties brought me in contact with model associations of Colorado and Wyoming, embracing near a thousand members, and representing a capital of hundreds of millions of dollars. These associations impressed me with the culture, marked cordiality, and enterprise of the frontiersmen. These gentlemen by resolve discourage the carrying of fire-arms, and the most intelligent superintendents are exemplary in their habits, and by precept and practice disallow the use of intoxicating drinks by their aids, while in the saddle or by camp fires in the "round-up." Good order is fast superseding a reign of violence, where it is no longer the fungi and parasites afflicting society, called outlaws, but factors and forces as men, to dignify and ornament American character.

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* The artesian wells, as a rule, are a signal success.

THE COW-BOY.

A wide-awake representative of 50,000 persons, equipped horsemen, as by story-tellers and in literary romance, is not to be judged a walking arsenal, the desperado, "sudden and quick in quarrel"; rather the tutor of romantic health-seeking youth from the east, who returns after a season, bronzed and robust; a skilled and courageous equestrian, the nation's trained cavalryman for an emergency, not a nomad of choice, often a partner in herd ownership, to become a valued citizen on the adoption of a new policy in land-leasing, checkering the domain with fences, supplanting the rude "shocks" by the spring with house and barn, in promise of greater comfort and rational content for the brute and his master.

In closing this report, I desire to express my hearty thanks to the honorable Commissioner for an assignment of an interesting field of inquiry under the new Bureau; also to make acknowledgment to gentlemen who have furnished information by personal interviews and correspondence. I cannot doubt that the expectations of enterprising and sagacious stockmen, and the voice of the late cattle conventions, held in Chicago and Saint Louis, will early be heeded and realized; that the Bureau of Animal Industry will diffuse needed information for the masses and formulate a just public opinion relative to the perils and losses to which our great industry is exposed. There is a popular demand for the inauguration of stern measures to stay the further ravages of contagious fevers and pleuro-pneumonia among our herds, kindred to those adopted in emergencies on the spread of fire or pestilence. Is it not practical—is it not the duty of our law-makers and servants by appropriations and skill to so far rid our country of contagious cattle diseases that at an early day we can make an honest proclamation to the world of the absolute health of our bovines? That will be the sign and signal for a revival of our animal industries; in the expansion of legitimate commerce, bringing larger gains to capital and labor, and in further promise of a national boast in the year 1890 that we, typical of our wealth and renown, will enumerate one hundred millions of cattle.

Very respectfully, &c.,

J. B. GRINNELL.

GRINNELL, IOWA, *December*, 1884.

LIVE STOCK AND MEAT TRAFFIC OF CHICAGO.

REPORT BY EDWARD W. PERRY.

Hon. GEORGE B. LORING,
Commissioner of Agriculture:

SIR: In a general sense the volume of the cattle traffic of the United States may be said to be shown by the receipts and shipments at five great receiving centers, Kansas City, Saint Louis, Chicago, Cincinnati, and Buffalo. Omaha has of late years been a receiving point, but nearly all cattle arriving there appear again in Chicago or other markets farther east. Previous to the end of the first quarter of the present century few droves of noteworthy magnitude were seen in the country west of Ohio, although some were driven from Pennsylvania and Ohio to the central part of Illinois. In the year 1825 the first passage of cattle through Chicago was noted, Col. William S. Hamilton having driven a herd from Springfield, Illinois, through Chicago to Green Bay, Wisconsin. Before the year 1833 Chicago had no provisions to export. As late as 1836 an actual scarcity of food created a panic among the people then living in that place. The first shipment of cattle products from what has become the greatest live-stock market in the world consisted of 287 barrels of salted beef and 14 barrels of tallow. That shipment was made by Newberry & Dole, on the schooner Napoleon, bound for Detroit. No trustworthy statistics relating to the cattle traffic of Chicago previous to the year 1851 were preserved, and from that year until 1856 no account of the receipts of cattle seem to have been kept. This was probably because a large number of those brought to Chicago were held on the open prairies until sold to butchers, to supply the requirements for local consumption. No count of the stock thus disposed of could well be obtained.

Beef packing was at that time, as for several years afterward, one of the chief industries of Chicago, and the record of the number of beeves salted there, from the year 1851 onward, appears to have been accurately kept, as was also the account of shipments of each year. All the statistics now obtainable relating to the annual receipts, shipment, packing, and consumption of beeves at Chicago, previous to the opening of the Chicago Union Stock-Yards, are presented in the subjoined table, together with the population of that city, year by year, and the number of beeves used per 1,000 of resident population. This estimate of the average consumption per 1,000 inhabitants has been used for calculating the receipts for the five years preceding 1856, but it can give at the best only a rough approximation. The number of cattle bought by

Chicago butchers, outside of the stock-yards, forms but a comparatively insignificant part of the entire supply required:

Years.	Receipts.	Shipments.	Packing.	Consumption.	Population.	Consumption per 1,000 population.
1851	*22,656	....	21,806	*850	*34,000	25.00
1852	*25,708	77	24,663	*968	38,734	25.00
1853	*29,908	2,657	25,431	*1,820	60,652	30.00
1854	*36,888	11,221	23,691	*1,976	65,872	30.00
1855	*39,865	8,253	28,972	*2,640	80,023	33.00
1856	39,950	22,205	14,971	2,774	84,113	33.02
1857	48,524	25,502	19,127	3,895	*93,000	41.88
1858	140,584	42,638	45,503	52,393	98,100	534.60
1859	111,694	87,584	51,606	22,504	*103,100	218.48
1860	177,101	97,474	34,623	45,004	109,206	412.89
1861	204,579	124,146	53,754	26,679	*113,000	236.09
1862	209,655	112,745	59,687	37,223	138,186	276.98
1863	300,622	187,048	70,086	43,488	*144,200	302.00
1864	303,726	162,446	92,459	48,821	169,353	288.81
Totals	1,691,410	833,996	566,379	291,035	†95,111	†218.19

* Estimated. † General average.

The above shows that the growth of the cattle traffic of the city named was very rapid during the first ten years. The shipments increased, in the decade ended with 1864, from 8,253 to 162,446, while in the same period the number of beeves packed rose from 28,972 to 92,459, an increase of 63,487. At that time one element of the success of Chicago as a market was the fact that stock might be pastured without charge on the prairies near the city, while the owners awaited a favorable condition of the markets in the Eastern States. The cattle were herded on the open prairies, buyers riding out to select such as seemed suited to their wants. The first stock-yards were located on the North Branch of the Chicago River, but they were used chiefly for swine. It was not until the year 1836 that the first cattle-yards were opened, a large tract near Twenty-ninth street, and east of Cottage Grove avenue, having been purchased, upon which a few stock pens were erected. The first scales for weighing live stock ever used in this locality were in those yards.

In 1865 the growth of the live-stock traffic led the several railroad companies at that time centering in Chicago, and some of the then managers of the stock-yards already existing, to combine for the erection of the Union Stock-Yards. These were opened for business on Christmas day, 1865, since which time, including the ten months ended with October 31, there have been received there 18,333,364 cattle and calves. Shipments of cattle during the same period were 11,795,490. This gives a total of 20,024,774 cattle received, and 12,629,486 shipped alive from the first records of the cattle trade of Chicago up to and including those made on the last day of October, 1884.

The subjoined table, showing the movement of cattle at the principal receiving markets of the country during the last twenty years, serves to illustrate step by step the growth of each of the several markets, and also exhibits their relative importance as slaughtering and as distributing points. The period included in the table began with 1865, the year in which occurred the opening of the Union Stock-Yards of Chicago and the ending of the civil war, which had disturbed greatly the course of the cattle traffic of the country. Kansas City sends to Chicago a considerable part of the cattle supply received, but not required for local consumption at the former place. Some of the native cattle, and a much larger percentage of the Texas stock which reaches Saint Louis, also go to Chicago. The greater part of the native cattle received at Saint Louis, however, go to markets in the Atlantic States, dealers in those markets, particularly those in New York, having agents stationed in Saint Louis to buy stock.

Movement of cattle at the principal markets of the United States during the nineteen years ended with December 31, 1883, and the ten months ended with October 31, 1884.

Years.	Kansas City.		Saint Louis.		Chicago.	
	Receipts.	Shipments.	Receipts.	Shipments.	Receipts.	Shipments.
1865	....	....	94,307	46,712	333,362	301,637
1866	....	....	103,259	24,462	393,007	263,608
1867	....	....	74,146	26,799	329,188	203,580
1868	....	....	115,352	37,277	324,524	215,987
1869	....	....	124,565	59,867	403,102	294,717
1870	....	....	201,422	129,748	532,964	391,709
1871	120,827	100,481	199,527	130,318	543,050	401,927
1872	236,802	206,467	263,404	164,870	648,075	510,025
1873	227,689	182,245	269,678	180,662	761,428	574,181
1874	207,080	166,519	360,925	226,678	843,966	822,929
1875	174,754	126,262	335,742	216,701	920,843	696,534
1876	183,378	120,346	349,043	220,430	1,096,745	797,724
1877	215,768	126,570	411,969	251,566	1,033,151	703,402
1878	175,314	131,761	406,325	261,066	1,083,068	699,108
1879	201,415	155,831	420,653	225,000	1,215,732	726,903
1880	244,709	199,421	424,720	228,879	1,382,477	886,614
1881	285,863	223,989	503,862	293,092	1,498,550	938,712
1882	439,671	359,012	443,169	188,486	1,582,530	921,009
1883	460,780	387,598	405,090	249,523	1,878,944	966,758
1884*	440,301	365,071	407,658	291,499	1,528,658	678,841
Totals	3,624,381	2,846,578	5,914,811	3,453,935	18,383,364	11,795,490

Years.	Cincinnati.		Buffalo.		New York.	Philadelphia.	Yearly totals.	
	Receipts.	Shipments.	Receipts.	Shipments.	Receipts.	Receipts.	Receipts.	Shipments.
1865	54,424	19,070	212,839	....	349,160	102,990	1,147,082	367,419
1866	79,503	31,300	275,091	....	371,965	111,330	1,334,155	319,455
1867	91,496	43,079	257,872	....	363,672	101,614	1,217,988	273,458
1868	87,459	43,315	265,101	267,892	369,023	99,714	1,261,173	564,471
1869	107,813	40,185	347,871	315,439	421,837	108,171	1,513,359	710,208
1870	107,167	54,681	388,057	361,017	475,068	126,738	1,831,416	937,155
1871	125,771	53,278	384,294	366,400	500,543	136,433	2,010,445	1,052,104
1872	169,855	76,866	379,086	346,635	551,759	247,102	2,496,083	1,304,868
1873	149,629	53,385	409,738	367,691	563,460	184,625	2,566,247	1,358,164
1874	190,426	79,551	504,594	468,921	562,428	185,140	2,863,559	1,564,598
1875	227,450	103,438	513,350	493,574	576,246	152,830	2,901,215	1,636,509
1876	243,503	98,322	615,790	500,139	589,464	191,550	3,269,473	1,826,961
1877	202,726	98,800	532,915	549,757	639,488	198,470	3,234,487	1,730,095
1878	182,276	87,181	657,809	604,784	682,057	188,600	3,375,479	1,784,500
1879	203,616	87,422	633,568	610,046	731,844	214,789	3,631,615	1,805,202
1880	229,451	81,189	786,386	668,520	857,632	218,606	4,143,981	2,059,623
1881	229,776	92,825	738,900	656,384	862,895	225,521	4,345,367	2,205,002
1882	241,671	96,014	630,955	595,408	831,400	230,120	4,399,516	2,159,929
1883	218,425	84,205	603,735	556,495	872,472	236,050	4,675,516	2,244,579
1884*	160,194	53,582	336,128	....	699,637	....	3,572,571	1,388,493
Totals	3,311,631	1,377,688	9,474,097	7,819,102	11,872,050	3,259,393	55,790,727	27,292,788

* For ten months ended with October 31, 1884.

The stock-yards of Kansas City, on the banks of the Kaw River, and just west of the boundary line between Missouri and Kansas, were first opened for business on June 1, 1871. Texas cattle formed by far the larger part of the receipts there then, as they do now, although the output of cattle from the States of Kansas and Colorado has grown greatly since that market was first opened. From that time to November 1, 1884, there were received in those yards 3,624,381 cattle, shipments in the period named having amounted to 2,846,473, leaving 787,908 for the local consumption of fourteen years, an average annual total of 56,279 cattle. From the opening of the Kansas City stock-yards the volume of traffic there was greater than was seen at any other markets during the first years of their existence. This was due to the fact that the annual drive of cattle from Texas to Kansas had been for some years established, and had reached a large volume before those yards were constructed. For several years almost the entire receipts of cattle at Kansas City were composed of Texas cattle. In time, however, the State of Kansas began sending forward considerable numbers of "native" cattle; later the ranges of Colorado, then of New Mexico, and finally of Arizona contributed to the receipts, as the Indians gradually retired from the ranges of the States and Territories named.

Of the cattle received at Kansas City previous to the erection of stock-yards for the common use of the several railroads then centering there, some were bought as early as 1868 for packing. In 1875 Nofsinger & Co. began the shipment of fresh beef in refrigerator cars, a business that has become an important branch of the live-stock traffic of Kansas City.

It is quite likely that the cattle market of Saint Louis had its beginning at least as early as that of Chicago, but it has not been deemed necessary for the purposes of this review to here carry this statistical account farther back than the year 1865. In the periods included in the above table it appears that while Kansas City has retained, for local consumption and for packing, only 21.5 per cent. of her total receipts of cattle, and Chicago has used 35.7 per cent., Saint Louis has shipped but 58.3 per cent. of her receipts, leaving 41.7 per cent. for home consumption. This is doubtless due to the fact that the population of Saint Louis has greatly exceeded that of Kansas City, while the receipts of live stock at the latter place were in 1871 only 78,700 less than those of Saint Louis. A noteworthy increase in receipts at Kansas City occurred in 1882, when 439,671 cattle arrived there. That number was only 3,498 less than the number of cattle received that year in Saint Louis. In 1883 receipts at Kansas City exceeded those of Saint Louis by 55,690 cattle. In the first ten months of the current year receipts at Saint Louis were 32,648 less than those at Kansas City during the same period.

SOURCES OF SUPPLY.

NATIVE CATTLE.

Previous to the beginning of the civil war, Saint Louis, Chicago, and other markets received some consignments of cattle from those States west of the Mississippi River, and south of the fortieth degree of latitude. The beginning of the war brought to an end that branch of the cattle traffic, and for years it was not revived. Despite this fact receipts in Chicago for 1860 were 5,407 greater than in 1859; in 1861 there was an increase of 87.478 over the next preceding year; in 1862 there was a further gain of 5,076, and in 1863 an increase of 90,967 over 1862, a total increase of 188,928 in receipts of the last-named year over 1859. This showed conclusively that the cattle industry of the region at that time tributary to Chicago was growing rapidly, without depending upon the Southwest for its increase.

The total yearly receipts of all the markets during the years ended with 1869 show this steady growth of the beef-producing industry more clearly, perhaps, than anything else can. The movement of cattle from Texas was small for a year or two next following the close of that war. Almost the entire supply of cattle at that time received at Chicago came from the States of Iowa, Missouri, Illinois, and Indiana. A few were derived from Wisconsin and from Michigan. Those from the last-named two States were then, as now, largely thin, unripe stock, intended for further feeding. The lighter among such animals are called "stockers," those of heavier weights and greater ages are called "feeders." Minnesota, Nebraska, and Kansas were new States then, requiring animals with which to stock farms and ranges rather than having a surplus of beeves. A limited number of bullocks were, however, sent eastward from those States.

Of the breeding of a large percentage of the supply of beeves received at the principal markets of the West very little definite knowledge was obtainable until quite recently. Such improvement in breeding as did appear among those beeves was, up to within the last five years, due almost entirely to the use of Short-horn sires. Of the so-called "grade" cattle now received, a large percentage show the influence of that race. Some cattle showing the characteristics of the Herefords have of late years appeared, as have also bullocks got by Aberdeen-Angus and by Galloway bulls. As the males got by pure-bred animals of the last-named three races are wanted for breeding purposes on the plains, the number sent to market is as yet comparatively small. About 75 per cent. of the beeves native to the Middle and Western States, which find their way to Chicago, have no defined characteristics of any established pure breed.

In the average weights and quality of the native beeves offered in the markets there has been a constant and marked improvement, due to some extent to the influences of improved sires. Much improvement has resulted also from better care and skill shown in feeding and sheltering the animals. In the earlier history of the West stock was quite generally without shelter, frequently half starved through the winter, and half fattened on wild grasses in summer. For water in the warm seasons they resorted to stagnant pools in the water-courses, to open ponds, with muddy shores, on the prairie, or to sluggish streams. When grain was fed it was almost invariably in the form of corn, unhusked and attached to the stalks. This was thrown upon the ground. If the latter was dry the cattle fattened fairly, but very often the fields were soft, poached until they were beds of mud, into which much of the grain and fodder was trampled by the stock. It is to the discredit of Illinois, Iowa, and Missouri that a like condition of affairs still exists on many farms. Even under the adverse influences of exposure, semi starvation, impure and insufficient water, and a wasteful mode of feeding, many of the beeves thrive, and at three or four years of age reach the market fairly fattened and of average weights, ranging from 1,100 to 1,400 pounds. Those to which superior care has been accorded attain a better condition, and average weights of 1,500 to 1,600 pounds. Cattle of greater average weights than the above have, almost without exception, some blood from some one of the improved races.

Those people who were engaged in the live-stock commission business in the Western markets twenty years ago say that the improvement of the general supply of native beeves from the introduction of good blood has been very marked, and that high-grade animals themselves are superior to those of like breeding seen twenty years ago. Cattle having in their veins half, three-quarters, or even larger percentages of the blood of purely-bred animals are shorter in the leg, broader in the back, thicker through the lung cavity, and generally more "blocky" than even the best offerings were a quarter of a century ago. It is quite certain that they mature at ages younger by some months than those at which steers usually ripened at the earlier period mentioned. Estimates as to the number of cattle improved by this introduction of the blood of established pure races show that in the nineteen States and Territories from which cattle are regularly sent to Chicago 16.5 per cent. are "grades," as those that have this admixture of pure and of common or "scrub" blood are called. In the seven corn-growing States, which find their chief market for beeves in Chicago, the percentage of high-grade cattle is .215 of the entire number.

A large number of thin steers, ranging in age from one to three years, are annually turned off by farmers in all of the States of the Upper Mississippi Valley. The new, the hilly, and the swampy parts of Michigan, Wisconsin, and Minnesota, especially, send numbers of animals of this description to market each year. In many cases half starved, thin, and seemingly stunted by the hardships of a life of browsing on swamp grasses in summer and a scant supply of hay, cornstalks, and brush in winter, these animals have digestive organs expanded by being stuffed with a great bulk of food containing little nutriment, and unimpaired by heavy feeding with rich grain. When placed in luxuriant pastures and fed a reasonable ration of grain, they make a very satisfactory growth, and at the age of three years reach a fair degree of maturity.

In the summer of the year 1880 a demand suddenly arose from farmers in Missouri, Iowa, and Illinois for young calves from the dairy regions of Michigan, Ohio, New York, and from other States of the seaboard. It is a curious fact that the purpose of buying such calves formed itself almost simultaneously in the minds of men living in widely separated parts of the West, and without communication or suggestion from each other upon the subject. The idea doubtless arose from the fact that pasturage in the Eastern States suffered extremely that year from the long-continued drought, while there was a scarcity of and strong demand for store cattle in the West. The movement of Eastern calves to the West grew with great rapidity during the first few months following its inception. Estimates made in the autumn of the year 1880 placed the number of such calves received at the Chicago stock-yards at 50,000, a number not beyond the limits of probability.

The experience of a few months showed that the risks attending the introduction of these calves were great. The prices paid therefor ranged from $8 to $20 per head. It was currently reported that the losses by death ranged from 20 to 90 per cent. of the various lots taken to Western farms. This statement was publicly and repeatedly made, and its truth does not appear to have been denied by any one having definite knowledge of the facts. These heavy losses apparently arose from the fact that many if not most of the calves were taken, without preparation for the change, from a diet of grass, milk, and such other soft and succulent food as is usually furnished to young calves. They were hurried to railway stations, crowded into cars, and kept for many hours upon their journey, receiving dry hay and cold water upon the way. Arrived at the stock-yards, they were placed in pens under roofs, where they were fed upon hay and water. Extreme hunger and thirst drove them to eat and drink. The water or other cause brought upon the tender young animals disorders of the bowels, resulting in speedy emaciation, and sometimes in death, before sale could be made.

When sold the calves were again placed in stock cars and taken to the neighborhoods where they were to remain. In some instances they were turned upon pasture where the grass was luxuriant, and died from diarrhea or other ailment. Others lingered, suffering from the attacks of lung-worms (*Strongylus micrurus*). Great numbers died of the latter cause, leaving scattered over the pastures the eggs of that parasite.

In the autumn of 1880 and afterward, farmers and others were repeatedly warned of the danger attending this branch of the cattle traffic, and especially of the risk incurred of introducing pleuro-pneumonia or other fatal contagious disease from the infected districts of the East. These warnings, echoed by live-stock and agricultural papers, had the effect of leading dealers to conceal from the public the sources whence came the calves they offered. Since those warnings were published by far the greater part of the supply received in the West has been accredited to Western New York and to Michigan. In 1881 the general assembly of the State of Illinois passed an act entitled "An act to suppress and prevent the spread of pleuro-pneumonia among cattle." This bill, approved May 31, 1884, was as follows:

AN ACT to suppress and prevent the spread of pleuro-pneumonia among cattle.

Be it enacted by the people of the State of Illinois, represented in the general assembly, That the governor of this State is hereby authorized and instructed to appoint a competent veterinary surgeon, who shall be known as State veterinarian or inspector, and whose duty it shall be to investigate any and all cases of contagious or infectious disease among domestic animals of the bovine species in this State which may be brought to his notice by a competent veterinary surgeon or practicing physician in the locality where such infectious or contagious disease may exist, and it shall be his duty to make visits of inspection to any locality where he may have reason to suspect that contagious or infectious disease may exist.

SEC. 2. In all cases of pleuro pneumonia among cattle in this State, the State veterinarian shall have authority to order the quarantine of infected premises, and in case such disease shall become epidemic in any locality in this State, the State veterinarian shall immediately notify the governor of the State, who shall thereupon issue his proclamation forbidding any animals of the kind among which said epidemic exists from being transported from said locality without a certificate from the State veterinarian showing such animals to be healthy. In case of epidemic, as aforesaid, the State veterinarian shall order the quarantine of infected premises, and shall order the slaughter of diseased animals thereon, and in cases of pleuro-pneumonia among cattle, he shall, as hereinafter provided, order the slaughter of all cattle upon the premises which have been exposed to contagion; but before doing so he shall call in consultation with him two reputable veterinarians or practicing physicians residing within ten miles of the infected premises, and shall not order the slaughter of any animals not actually diseased without a written order signed by one or both of the said veterinarians or practicing physicians.

SEC. 3. Whenever it becomes necessary, as herein provided, to order the slaughter of animals, the State veterinarian shall notify the nearest justice of the peace, who shall thereupon summons three disinterested freeholders of the neighborhood as appraisers of the value of such animals. Said appraisers, before entering upon the discharge of their duty, shall be sworn to make a true and faithful appraisement, without prejudice or favor. They shall, after making their appraisement, return a certified copy of their valuation to the justice of the peace by whom they were summoned, who shall, after entering the same upon his record, and making an indorsement thereon, showing the same to be properly recorded, return it, together with the order of the State veterinarian, to the person or persons owing live stock ordered slaughtered.

SEC. 4. Whenever the governor of the State shall have good reason to believe that such disease has become epidemic in certain localities in other States, or that there are conditions which render such domestic animals liable to convey disease, he shall thereupon, by proclamation, schedule such localities and prohibit the importation of any live stock of the kind diseased into this State, unless accompanied by a certificate of health, properly signed by a duly authorized veterinary inspector. Any corporation or individual who shall transport, receive, or convey such prohibited stock shall be deemed guilty of a misdemeanor, and upon conviction thereof shall be fined not less than one thousand dollars or more than ten thousand dollars for each and every offense, and shall become liable for any and all damage or loss that may be sustained by any party or parties by reason of the importation or transportation of such prohibited stock.

SEC. 5. If any person or persons shall have upon his premises any case of pleuro-pneumonia among cattle, and shall fail to immediately report the same to the State veterinarian, or if any person or persons shall willfully and maliciously obstruct or resist the State veterinarian in the discharge of his duty, as herein before set forth, he shall be deemed guilty of a misdemeanor, and, upon conviction of either charge, shall be fined not less than fifty dollars nor more than five hundred dollars for each and every such offense, and upon conviction a second time shall, in addition to the above-named fine, be liable to not less than thirty days nor more than six months imprisonment.

SEC. 6. The State veterinarian shall annually make a report to the governor of all matters connected with his work, and the governor shall transmit to the department of agriculture such parts of said report as may be of general interest to breeders of live stock, to be published with the proceedings of the State board of agriculture.

SEC. 7. All claims against the State arising from the slaughter of animals, as herein provided for, shall, together with the order of the State veterinarian, and the award of the appraisers in each case, be submitted to the governor, and he shall, after having examined each case, if satisfied of the justice of the same, indorse thereon his order to the State auditor, who shall thereupon issue his warrant on the State treasurer for the same so ordered paid by the governor.

SEC. 8. The State veterinarian shall be entitled to receive for his services the sum of eight dollars per day for every day actually employed under the provisions of this act, together with his necessary traveling expenses. He shall make an itemized account to the governor, properly signed and sworn to, of the number of days he has served, and of the expenses which he has paid, and the governor shall, if satisfied that the same is right and proper, indorse thereon his order on the State auditor for the amount. The appraisers, heretofore provided for, shall be entitled to receive the sum of one dollar each for their services, to be paid out of the treasury of their respective counties, upon certificate of the justice of the peace summoning them. The justice of the peace shall be entitled to receive the ordinary fee for issuing summons, to be paid out of the town in counties not under township organization, and out of the county fund in counties not under township organization. The physicians called in consultation shall be entitled to receive for their services the sum of two dollars per day, and mileage at the rate of ten cents per mile one way; such compensation and mileage to be paid out of the veterinarian contingent fund. The State veterinarian shall have at his disposition the sum of two thousand dollars, to be expended in disinfecting infected premises and other incidental expenses connected with his work, for which he shall, before entering upon the discharge of his duties, give bond, with good and sufficient security, in the sum of five thousand dollars, and shall make a sworn statement to the governor of the amount he disburses. Any part of said two thousand dollars not used shall lapse into the State treasury.

SEC. 9. For the purpose of carrying out the provisions of this act, the sum of eight thousand dollars, or so much thereof as is necessary, is hereby appropriated out of the State treasury, to be paid, as hereby provided, out of any funds not otherwise appropriated.

In response to solicitations of a large number of owners of live stock, and upon evidence furnished by them, Hon. S. M. Cullom, then governor of the State of Illinois, issued the proclamation of which the subjoined is a copy:

STATE OF ILLINOIS, EXECUTIVE DEPARTMENT,
Springfield, November 1, 1881.

In pursuance of the act of the general assembly of Illinois, entitled "An act to suppress and prevent the spread of pleuro-pneumonia among cattle," approved May 31, 1881, I, Shelby M. Cullom, governor of the State of Illinois, do hereby proclaim that I have good reason to believe that pleuro-pneumonia among cattle has become epidemic in certain localities in the States of Conecticut, New York, Pennsylvania, New Jersey, Delaware, and Maryland, viz: In the county of Fairfield, in the State of Connecticut; in the counties of Putnam, Westchester, New York, Kings, and Queens, in the State of New York; in the counties of Lehigh, Bucks, Berks, Montgomery, Philadelphia, Delaware, Chester, Lancaster, York, Adams, and Cumber and, in the State of Pennsylvania; in the counties of Bergen, Hudson, Morris, Essex, Union, Somerset, Hunterdon, Middlesex, Mercer, Monmouth, Ocean, Burlington, Camden, Gloucester, and Atlantic, in the State of New Jersey; in the county of New Castle, in the State of Delaware, and in the counties of Cecil, Harford, Baltimore, Howard, and Carroll, in the State of Maryland; and I do hereby, as required by said act, prohibit the importation of any domestic animals of the bovine species into this State from the aforesaid counties, in the States of Connecticut, New York, Pennsylvania, New

Jersey, Delaware, and Maryland, after the 10th day of November, instant, unless accompanied by a certificate of health, properly signed by a duly authorized veterinary inspector.

"Any corporation or individual who shall transport, receive, or convey such prohibited stock shall be deemed guilty of a misdemeanor, and, upon conviction thereof, shall be fined not less than $1,000 nor more than $10,000 for each and every offense, and shall be liable for any and all damage or loss that may be sustained by any party or parties by reason of the importation or transportation of such prohibited stock." (Sec. 4 of act approved May 31, 1881.)

In testimony whereof I hereto set my hand and cause the great seal of the State to be affixed. Done at the city of Springfield, the day and year above written.

S. M. CULLOM.

By the Governor:
HENRY D. DEMENT, *Secretary of State.*

Copies of the above act and proclamation were sent to the managers of the railroads connecting Chicago with the States wherein there was reason to believe contagious pleuro-pneumonia existed. Both act and proclamation were widely published, and the penalty for violation of the provisions of the act thus became quite generally known. The effect is fully shown by the tables which are given herewith, from which it appears that receipts of calves were 23,931 less in the year next following the prohibition of importations from scheduled districts than they were in 1881. A part of that falling off in receipts, however, may have resulted from the fact that in 1882 the conditions of the pastures in the East were more favorable to the raising of the calves there than they were when the traffic began, while the demand in the West was much less urgent, owing to the fears excited by the warnings of the friends of the cattle business of the country.

In the subjoined table are given figures showing the movement of calves in the Chicago market, during the years in which accounts of receipts and shipments thereof have been kept separate from those of other cattle:

Railroads.	1881.		1882.		1883.		* 1884.		Totals.	
	Receipts.	Shipments.	Receipts.	Shipments.	Receipts.	Shipments.	Receipts.	Shipments.	Receipts.	Shipments.
Baltimore and Ohio	9,060	50	853	8	835	178	2,170	107	12,418	838
Chicago and Alton	269	1,228	206	837	164	964	232	2,684	961	5,713
Chicago and Atlantic	......	......	......	......	1,223	2	8,536	1	9,759	3
Chicago, Burlington and Quincy	1,023	9,854	2,174	1,678	1,541	1,862	611	8,925	5,349	22,319
Chicago and Eastern Illinois	804	486	636	212	515	96	46	362	2,422	1,156
Chicago and Grand Trunk	4,011	......	802	40	996	4	1,896	15	7,705	59
Chicago, Milwaukee and Saint Paul	4,339	1,099	4,067	1,082	8,456	1,530	6,675	1,780	23,537	5,491
Chicago and Northwestern	4,885	4,425	5,420	1,795	6,511	1,924	5,423	1,883	22,237	10,027
Chicago, Rock Island and Pacific	248	10,815	601	1,342	576	1,821	248	4,987	1,673	18,965
† Chicago, Saint Louis and Pittsburgh	1,230	146	373	84	189	134	484	226	2,276	590
Illinois Central	1,270	2,225	967	1,410	1,392	1,284	931	2,247	4,560	7,166
Lake Shore and Michigan Southern	13,990	11	4,527	831	2,929	304	10,004	43	31,450	1,189
Louisville, New Albany and Chicago	......	......	388	139	343	144	756	101	1,487	384
Michigan Central	5,666	98	1,549	28	2,478	131	3,845	5	13,538	262
New York, Chicago and Saint Louis	......	......	3	......	710	513	686	192	1,399	705
Pittsburgh, Fort Wayne and Chicago	1,736	......	1,834	75	1,335	42	1,257	57	6,162	174
Wabash, Saint Louis and Pacific	263	3,028	373	673	479	1,238	224	1,944	1,339	6,883
Yearly totals	48,794	33,465	24,863	10,229	30,172	12,171	44,445	25,569	148,354	81,124

* For ten months ended with October 31. † Formerly Pittsburgh, Cincinnati and Saint Louis R. R.

The above table shows that in 1882 receipts of calves were 466 more than one-half of the number received in 1881. In 1883 receipts rose to 30,172, and in the first ten months of 1884 they were 44,445. The increase and decrease in receipts in 1882 over those of 1881, in 1883 over 1882, and in the first ten months of 1884 over those of the year 1883 are shown in detail below, as divided among the several railroads:

Railroads.	1882.		1883.		1884.	
	Increase.	Decrease.	Increase.	Decrease.	Increase.	Decrease.
Baltimore and Ohio		8,207		518	1,835	
Chicago and Alton	27			132	68	
Chicago and Atlantic			1,223		7,813	
Chicago, Burlington and Quincy	1,151			633		930
Chicago and Eastern Illinois		168		121		48
Chicago and Grand Trunk		3,209	104		900	
Chicago, Milwaukee and Saint Paul		272	4,389			1,781
Chicago and Northwestern	535		1,091			1,088
Chicago, Rock Island and Pacific	353			25		328
Chicago, Saint Louis and Pittsburgh		857		184	295	
Illinois Central		303	425			461
Lake Shore and Michigan Southern		9,463		1,598	7,075	
Louisville, New Albany and Chicago	388			45	413	
Michigan Central		4,117	929		1,367	
New York, Chicago and Saint Louis	3		707			24
Pittsburgh, Fort Wayne and Chicago	98			499		78
Wabash, Saint Louis and Pacific	110		106			255
Totals	2,665	26,596	9,064	3,755	19,266	4,993

A summary of the above, showing, in a general way, the increase and the decrease, year by year, in the movement from Eastern and from Western sources is subjoined:

Sources.	1882.		1883.		1884.	
	Increase.	Decrease.	Increase.	Decrease.	Increase.	Decrease.
Eastern railroads	489	26,293	3,053	2,965	19,198	150
Western railroads	2,176	303	6,011	790	68	4,843
Totals	2,665	26,596	9,064	3,755	19,266	4,993

For some reason, the movement from the East has this year recovered its full vigor. If receipts for November and December shall prove as heavy as the average for the first ten months of the current year, the total for the year, over Eastern railroads, will be nearly 44,000, and exceed those by the roads mentioned for any previous year by some 7,500.

TEXAS CATTLE.

The history of the cattle native to the plains of Texas takes the inquirer back to the times of the invasion of that region by the Spaniards. From cattle that escaped from those brought over by Cortez, the Spanish conqueror, or were abandoned by his followers, have descended the hundreds of thousands of animals strongly resembling those of Andalusia in their bony form, their slender, long legs, light flanks, and large, wide-spreading horns. For many generations they were neglected and multiplied, forming no part of the food supply of the people of the United States.

The Mississippi River was for a time a favorite route with shippers of cattle from Texas; but before the beginning of the late civil war herds were driven through the Indian Territory to Missouri and Kansas. Early in the history of that movement the malady known as Texas fever killed cattle that grazed on lands over which the herds from Texas passed. Great indignation was aroused among the people living in the regions traversed by the southern cattle, and armed parties sometimes seized those having the traveling stock in charge, and by threats, and occasionally by flogging or other outrages, turned them from their intended route. Sometimes the herds were scattered and never again brought together.

As early as the year 1858, herds of these cattle from the Southwest were driven to Chicago. Two years later this traffic was cut off entirely, and until after the fall of Richmond few cattle from Texas found their way to the North. During the war in the South stock from Texas was driven through Louisiana to supply the Confederate armies east of the Mississippi. New Orleans and Old Mexico afforded a market for a limited number of cattle, but Kansas offered opportunity, four or five years after the close of the war, for much larger sales.

The characteristics of the native cattle of Texas are in marked contrast with those of the established pure breeds of beef animals, but there is a slight resemblance in form and color to some dairy cattle, particularly those of the Channel Islands, although the Texas cow lacks that development of the hinder parts that denotes a capacity for milk production. Usually the cattle of Texas are a yellow dun in color. Their bones are slender and covered with little flesh and less fat. The hind quarters are light, the backbone is prominent, the hip or hookbones are high and narrow, the ribs comparatively straight, the chest narrow, the head bony and large, and the horns almost invariably long and sharp, while they are frequently enormous in diameter and in spread. Yet, upon good rations, the animals fatten rapidly to a reasonable degree, and their flesh is sometimes tender, juicy, and fairly marbled throughout, while it is almost without exception apparently healthy. Many thousands of these animals are driven northward from Texas each year, the average annual drive being a little more than a quarter of a million. The younger cattle go to the grazing grounds of Colorado, Wyoming, Dakota, and Nebraska to spend one or two years. They are then sent to the corn-producing States to be fattened, or are shipped direct to market, usually to Chicago. Others are sent direct from the towns on the Atchison, Topeka and Santa Fé, or other railroads in Kansas, to the great markets, while yet others are sold in Kansas to the farmers of that State and Nebraska. The latter pasture the stock in summer, and either feed hay or corn during the first winter, and sell the cattle when ready for market.

The average weights of those cattle which have been brought direct from Texas, technically known as "through," or as "grass Texans," range from 650 to 950 pounds. Large numbers of such animals are bought by those who slaughter beeves for shipment in the dressed form, or for preserving in tin cases or cans. For the latter purpose the Texas stock is perhaps as good as, if not better, than any other found in this country. While the flesh is not usually tough and stringy, as that of old domestic cattle is likely to be, it is free from an excess of fat. As the latter is objectionable in canned beef, and as the price of tallow is much lower than that of nutritious flesh, the absence of any superabundance of fat is greatly to the advantage of those who use the cattle for the purpose of preserving their flesh in cans.

WESTERN CATTLE.

The plains of Western Kansas and Nebraska, and the region lying between those States and California and Oregon, form a source whence Chicago receives a supply of cattle rapidly increasing in numbers as well as improving in quality. The herds of those regions have descended from two distinct sources. The chief of these was the grazing grounds of Texas, which have for almost twenty years contributed large numbers of steers for grazing purposes, as well as many cows for breeding, to the ranches of the West and Northwest. Another source of supply was the States east of the Missouri. Cows and heifers in considerable numbers have been taken to the plains from these States, the movement in this direction having been especially heavy in the year 1883. On the plains these females were coupled with pure-bred bulls, or to high-grade males, got by pure-bred Shorthorn, Hereford, Galloway, or by Aberdeen-Angus bulls. In number the males of the races mentioned rank on the plains in the order in which they are named, the number of Shorthorn bulls exceeding all others of pure blood. In Colorado and nine of the principal cattle-growing Territories there are 416,890 high-grade cattle among the entire supply of 4,299,559 cattle in those Territories and that State. The first cattle marketed from the West, showing the influence of the Aberdeen-Angus blood, appeared in the Chicago stock-yards in 1879. Since that time a few have appeared from time to time, usually in small numbers. Males having 50 per cent. or more of the blood of any of the races named above, excepting the Shorthorn, are usually kept on the range for breeding purposes. Females of that description are invariably kept for that purpose. Thus a very marked improvement in the quality, weight, and value of the supply of cattle on the ranges is making. But this change is by no means fully shown by a corresponding change in those sent to the markets for beeves. It is quite likely that some five or six years will pass before a large part of the output of the plains will show this improvement.

In weight the unimproved cattle of the plains range from 900 to 1,000 pounds. In value they are usually from 50 cents to $1 per cental above Texas cattle of like age and weights, because they are, as a rule, more fleshy. They fatten readily upon grain, and large numbers are thus fattened in the corn-producing States of the West each year. Half-breed animals from the plains, grown under conditions the same as those surrounding the unimproved stock above mentioned, range in weight from 1,000 to 1,250 when in moderate condition, and bring from $1 to $1.50 per cental more in the market, for beeves or as store cattle, than do the inferior cattle. The "grades" fatten much more readily than do the others and their flesh is said to be of much better quality than that of the unimproved animals.

These Western cattle are considered by the slaughterers for shipment in carcasses, and also for canning, as being superior to any other cattle offered in the market, since they are not excessively fat, but are well fleshed, healthy, and their meat is of good quality.

TRANSPORTATION.

Only two means for transportation of cattle to Chicago are now commonly used in any part of the routes from their native homes to the great central markets. The cattle are driven to some one of the seventeen railroads reaching Chicago, or to their branches. The longest of these drives is from the grazing grounds of the Gulf coast to the railroads in Kansas or or in Nebraska. While on that drive the stock move at the rate of some 15 miles per day, starting early in the year, as soon as the grass along the way has grown enough to afford them sustenance. They usually arrive at the railroad in an emaciated condition. There the stock are held in the neighborhood of shipping stations until satisfactory sale can be made to buyers at those shipping points or until the owner sees fit to ship to market. From sixteen to twenty animals of heavy weights, or twenty-two of the lighter weights, of cattle are crowded into each car. Stock cars were, until a few years ago, 28 feet long and 8 feet wide; but with the improvement in road-beds, the use of heavier and better rails and stronger and more serviceable locomotives and trucks, cattle cars have been enlarged from time to time, until many of them now are 34 feet long and 8 feet 6 inches to 8 feet 8 inches wide. Some years ago railroad companies refused to haul more than 20,000 pounds in any freight car. For years this constituted a standard car-load. Later the shipper was allowed to put 24,000 pounds in each car, but was charged a much higher rate per cental for the excess over 20,000 than the rate per cental for a standard car load. In later years the freight tariff has been based upon the actual weight of the articles carried, and the charge per car-load has been to a considerable extent abandoned, except in the case of live stock. Railroad officials are now considering the advisability of fixing rates upon live stock, as upon dead freight, at a given amount per cental. It is held by those favoring this plan that a charge based upon the actual weight would tend strongly to put a stop to overcrowding stock-cars, and would thus save much suffering among animals in transit, as well as a considerable loss to the shipper, which results from that of overloading.

One of the chief causes of the enlargement of freight cars was the scarcity of rolling stock of that kind. It was found that while much more freight could be placed in one of these larger cars, an engine could move a given number of them almost as easily as it could move a like number of smaller cars, carrying a lighter load. Another reason for the enlargement was found in the fact that track room, particularly in the larger cities, had become so valuable as to require the use of all practicable means for economizing space. Wider, longer and stronger cars, carrying from 40,000 to 45,000 pounds, are now in common use, while on at least one road in the West cars capable of safely carrying 70,000 pounds, or the equivalent of three and one-half of the old standard car-loads, are seen. Such, however, have not yet been used for the transportation of beeves.

At nearly every station on the lines of the railroads centering in the live-stock markets of the West, stock-yards are provided for the accommodation of shippers. Each of these yards has one or more inclined platforms, rising from the ground to the height of the floor of stock-cars, and guarded on each side by strong fences. When ready for shipment the cattle are hurried with shouts and blows into the narrow "shute," as the ascending platform is called. As soon as all the cattle to be shipped in the car then before the shute are in the latter, a gate is closed behind them. They are then urged onward by loud cries, by blows, and by thrusts of poles in the ends of which sharp-pointed brads or nails are fixed. As the shute is too narrow to admit of the passage of two beeves side by side, there is but one way open to the latter, and that is through the open door in the side of the car. Into this the cattle crowd until the car is packed to its utmost capacity. When thus loaded, cattle cars are so full that when once down an animal finds it scarcely possible to rise, but lies beneath its fellows, not infrequently becoming so bruised by their trampling hoofs as to be unable to stand unsupported or rise to its feet while on the way or when the car is unloaded at the feeding stations or in the general market. To prevent losses from this cause, men accompany the trains of cattle for the purpose of rousing to their feet such animals as may get down. It is claimed by shippers that crowding the cars to their utmost capacity tends to keep the cattle upon their feet, since when thus crowded in they support each other. An ordinary car will hold, when thus crowded, fourteen or fifteen fully matured beeves of good quality, averaging about 1,600 pounds, making a car-load of 22,000 to 24,000 pounds. Since the freight charge is by the car-load, instead of by the cental, it is probable that the desire of the shipper to lessen the average cost of transportation per bullock is the real reason for loading the cars so fully. It is likely, therefore, that if the railroad companies shall decide to adopt the plan of charging by the hundred pounds for the transportation of all live stock, the effect will be to lessen the suffering of animals in transit, since there would then be no special gain in overloading cars. Under the present system not a train of cattle is brought to any great market without having many crippled beeves, and not infrequently several dead ones, which must be sold at a price but little above the value of the hide to the rendering companies, to which, under the statutes of the several States in which the great markets are located, all dead animals must be delivered. In Chicago the price for dead cattle ranges from $5 to $10 for bullocks that might be worth alive from $40 to $80 each.

Accompanying each train of cattle are men whose duty it is to see that the stock is properly cared for in transit, receiving water and food at the proper intervals, and to rouse those animals which sink under the hardships of the journey and are likely to become trampled under the feet of their companions. The means used by these men for thus rousing cattle that lie down were in the past much more cruel than those now adopted. The change is largely due to the efforts of the several humane societies, which have vigorously prosecuted offenders against the laws passed for the protection of dumb beasts from cruelty. At present the instruments used, particularly in the State of Illinois, where the efforts of the Illinois Humane Society have been very vigorous consist of poles in one end of each of which has been driven a nail, sharpened to a point, that extends from one-quarter to one-half an inch beyond the wood. With this the cattle are pricked until they arise. Even with this comparatively harmless instrument many cattle are tortured severely.

Once placed in cars, cattle are kept on the way toward market for periods ranging from fifteen to twenty-four hours, without unloading for rest, food, or water. In some instances longer periods pass without unloading the stock, particularly when, for any cause, stock trains are delayed on the road. Railroad managers strive to facilitate the movement of stock, particularly of cattle trains, as much as possible, and on some roads tributary to Chicago the time made by stock trains is but little slower than that of passenger trains.

Arrived at the Chicago stock-yards each train is placed beside a platform, even in height with the floors of the cars, and about 8 inches distant therefrom. As soon as the door of the car is opened this space between car and platform is covered by a piece of plank equal in length to the width of the doors of the car, and held in place by two strong pieces of iron resting upon the platform at one end and upon the floor of the car at the other. Gates long enough to extend across the width of the platform are then so placed as to inclose a space equal in length to that of a stock car, and the cattle are driven upon the platform. Thence they find their way down an incline to a pen large enough to hold from two to three car-loads of cattle. Any crippled or dead animals in the cars are then dragged to the platform, and are usually left there for a time, unless there appears to be reason for believing that the cripples will struggle to their feet and stray away.

The platforms used for unloading stock extend, practically, entirely around the stock-yards. Those on the north side are used by the Pittsburgh, Fort Wayne and Chicago, the Lake Shore and Michigan Southern, the Illinois Central, and the Chicago, Burlington and Quincy Railroads. Those on the west, by the Chicago and Alton, the Chicago and Northwestern, the Chicago, Milwaukee and Saint Paul, the Chicago and Eastern Illinois, and the Wabash, Saint Louis and Pacific Railways. Those on the east, by the Chicago, Rock Island and Pacific, the Baltimore and Ohio, and the Chicago and Grand Trunk Railways. Shipments over the Lake Shore and Michigan Southern and the Pittsburgh, Fort Wayne and Chicago Railways are made from Division B at the north side of the stock-yards.

The Chicago Union Stock-yards practically occupy an area of 280 acres extending from Halstead street on the east to Packers avenue on the west, and from Transit avenue southward to Forty-seventh street. Of this area a part off the west is occupied by packing houses and slaughtering establishments. On the east are the Transit House, a hotel owned by the Union Stock-Yard and Transit Company; large stables, a printing office, post-office, stations for railway and street-car lines, and, south of all these, buildings provided for the accommodation of those who wish to hold public sales of fine cattle. During the current year a building was erected on these grounds to shelter those in attendance at such public sales. For the purposes of the traffic of the stock-yards some fifty miles of heavy steel railway track have been laid and are in almost constant use. These lines connect with every railway entering Chicago in such a manner that it is not necessary for any live-stock destined for the market to enter the city. Not far from the railway and street-car stations, and within the limits of the yard proper, is an exchange building, in which the commission merchants, the Union Stock-Yard and Transit Company, a telegraph, a telephone, and some railroad companies have offices. In an annex to this building is the Union Stock-Yard National Bank, and in another adjoining structure is a large dining room. A short distance from the exchange building is a station for the fire department of the Union Stock-Yard and Transit Company, in which a steam fire-engine is kept.

In front of the exchange building, and 500 feet distant, stands a water tower 135 feet high, in which is a stand-pipe 7 feet in diameter. Near it is an engine-house containing large pumps, which force into the stand-pipe water drawn from three artesian wells. These wells furnish a constant supply of water great enough at all times for the wants of 100,000 swine and 20,000 cattle. A large part of the yards consist of open pens, but the divisions allotted to swine and sheep are covered by roofs, the lower parts of which are about 8 feet from the flooring, which extends under all of these covered pens. Many of the uncovered cattle-pens are also floored, heavy plank being used for this purpose. These floors are 1 to 2 feet above the surface of the ground, thus keeping the cattle above all mud and water. Under these floors large open drains extend to carry off any rainfall and waste from the water pipes. This drainage, together with that from the packing-houses west of the yard, empties into the south branch of the Chicago River. In each division of the yard scales are provided for weighing the stock. Viaducts are also provided over which swine and sheep are driven, high above the cattle in the pens beneath. These viaducts are commonly used as a passage-way for swine to the packing-houses. Cattle are usually driven through the alleys and streets of the yards to the shipping-pens, or to slaughter-houses adjacent to the yards, or in the city. A viaduct for teams extends from east to west across the yards south of Dexter Park.

A walk for pedestrians extends from Halstead street on the east, along the south side of Center avenue, to the western limits of the yards. For the convenience of operators, a plank is fixed on the top of the fences surrounding the pens. This plank is about 12 inches wide, and serves as a walk from which buyers and sellers can survey the stock in the pens. In the divisions where swine are sold, streets, alleys, and pens are all floored; but among the cattle pens the streets and alleys are generally filled with cinders or slag from the iron works at Bridgeport, making a firm and comparatively dry passage-way in all weathers.

From the pens beside the platforms cattle are counted out by the employés of the Stock Yard Company, and are then under the care of the said company, until an order is given by the consignee for their delivery to the purchaser, or, in event of shipment to other markets, an order for their delivery to some railroad. Each pen is kept locked by the Stock-Yard Company; but a large number of the commission merchants are furnished with keys to the locks, and can transfer at their own will cattle consigned to them. Before getting these keys, the commission merchants furnish a bond to the Stock-Yard Company sufficient to cover any losses which may arise through carelessness, or through improper use of such keys. As soon as convenient after the stock is placed in the pens into which the stock-yards are divided, hay is furnished. After ample time has been given for eating the hay, troughs fixed in each pen are filled with water and the cattle allowed to drink. They are then ready for weighing to the buyer. In the mean time the salesman will have shown the cattle to such buyers as may be dealing in stock of the class he may have to offer.

Cattle consigned to the live-stock markets of the West are, as a rule, sold by weight. Salesmen charge a commission, usually 50 cents per head, for their services. These consist of ordering hay for the stock as soon as possible after the animals have been yarded in the allotted pens; of supplying the animals with an abundance of pure water, after they have eaten all the hay extreme hunger can induce them to consume; of offering the stock to all dealers likely to buy; of paying all charges of railroad and stock-yard companies for freight, yardarge, or for hay; of collecting payment for stock sold; and of rendering to the consignee an account of the whole. This requires that the salesman or his representative shall be in the stock-yards at an early hour in the morning, frequently long before dawn. He must also often stay until a late hour in the evening to receive consignments. His work demands his personal attention at all hours of the day, and in stormy as well as in pleasant weather. When the worst storms are raging he must be busiest, looking after the welfare of the stock sent to him. It may be truly said that at no time is the work of the live-stock salesman agreeable, for when the ground is wet he must splash through the mud everywhere present in the yards; and in dry weather he cannot avoid the clouds of dust whirled by every passing breeze through streets and alleys.

It has long been a quite general custom for consignors of stock to draw upon the consignee for a part, at least, of the purchase price of animals shipped to the latter. Usually the draft, accompanied frequently, if not invariably, by a bill of lading, reaches the consignee before the stock on account of which the draft is made arrives at the yards. To meet such cases the live-stock broker finds it necessary to have a good standing with the bank in the stock-yards, where all the banking connected directly with the business of the market is done. It will be seen that the consignee needs but a comparatively small cash capital with which to carry on the business of buying and shipping cattle, since he can obtain upon his drafts on his commission merchant nearly or quite as much as he pays for the stock he ships.

The custom of drawing against the amount it is expected the cattle will bring in the market has been served to make it almost the invariable practice to sell stock on the day of its arrival, whenever this is possible. However much the salesman may believe more money could be obtained for the stock by holding it for the market of another day, the fact that he has already made a large advance upon the cattle leads him to sell, that he may get the money with which to replace that advanced. The other reasons for disposing of all consignments on the day of their arrival are that the expense of feeding is very great, since the price charged for hay is high, and that the salesman realizes that should he hold the stock and be compelled to accept a price no higher than he could have obtained on the day of the arrival, the consignor will have ground for complaint. By common consent the market for cattle ends at 3 o'clock p. m., at which time a whistle on the engine-house beside the water-tank is blown, to notify dealers that the market hours of the day are ended.

There are several distinct branches or divisions of the cattle market at Chicago, each having characteristics peculiar to itself. Chief among these, and a constant feature of the trade at all seasons, is that of the higher classes of "native" cattle. Of these the best are termed "export" cattle, because they are of the kind chosen by those who buy for exportation alive to England. Next to these come native cattle of inferior quality, but intended for immediate slaughter. Still less matured bullocks are called "feeders," and next below the latter come "stockers," the last two classes forming the supply from which graziers and feeders of stock obtain the store cattle they wish to fatten. If the intention is to put the stock upon a diet of grain at once, the buyer selects the heavier animals known as "feeders." These weigh from 900 to 1,100 pounds each, and are usually two or three years old, having a large frame and comparatively little flesh. "Stockers" are light young cattle, frequently yearlings, and range in weight from 500 to 800 pounds each. They are turned into stalk-fields, or fed upon corn-stalks, hay, or straw during one winter, go into pasture the next summer, and are, as a rule, then put upon full rations, to be finished for market. "Butcher stock" consists of thin, rough steers, skinny old cows, bulls, oxen, and the refuse generally of the cattle market. They are usually bought to supply the home demand of the city of Chicago. Milch-cows and "springers" complete the list of so-called "native" cattle sold in the Chicago market. For the accommodation of dealers in the last-named class, comfortable and ample sheds have been provided. In the Chicago market are a large number of men who have long been termed "scalpers," a name applied generally to those who buy cattle or swine of the commission merchants, for the purpose of selling again in the market here. They select from the consignments of stock such animals as will suit their own customers, and not infrequently some of the larger operators will have hundreds of animals in the yards, day after day, awaiting buyers. From these cattle each purchaser may select such as seem mostly likely to suit his purpose, the entire supply being disposed of usually to farmers; but sometimes those rejected by purchasers who wish to fatten the stock are sold to local butchers, and enter into the supply for city consumption.

It is these "stockers" and "feeders" that receive in the stock yards, at certain seasons of the year, the germs of the disease known as Texas fever, and afterward die of that malady when taken to the country. Texas and Western cattle each form distinct yet kindred branches of the cattle market of Chicago, differing from those mentioned above in that the cattle from the plains are usually sold in large lots to those who ship dressed beef, or to packers and canners who do business in Chicago.

BEEF-CANNING.

As long ago as the year 1810 meats were preserved by processes patented in England by one Appert. During the late civil war in this country large quantities of beef preserved by methods similar to, if not identical with, those covered by the Appert patent were used in the United States Navy. Most, if not all, of this beef was put up in the cities of Portland, Me., and Boston, Mass., under patents issued to Mr. Hezekiah Winslow. Meats thus preserved were placed in a raw state in cans which were then put into vats and thoroughly cooked, the cans being sealed immediately after the cooking. Thus treated the contents of the cans consisted of a considerable quantity of fluid in which the meat was suspended loosely. The flavor of beef thus preserved was not very agreeable, as it seemed to have been overcooked, and tasted as though it had been scorched. A large part of the meat preserved in cans in Australia and in New Zealand resembles that above described, being put up in round tin cases, or "tins," as they are commonly called in England and her colonies.

On March 31, 1874, the business of putting up compressed cooked beef in tin cans of pyramidal form began in Chicago. The Wilson Packing Company was the first firm or corporation of any kind that ever packed compressed beef, so called, in cans of that shape. Mr. William J. Wilson, senior partner of the firm named, introduced these goods in the New York market by carrying samples around in a basket, and showing their merits to the trade. The business was very discouraging for a time, many not hesitating to say that Mr. Wilson was a lunatic. From that seemingly insignificant and unpromising beginning the trade in canned meats has extended to the markets of every nation, and it is evident that in the opinion of people of all lands where such meats have been introduced, the goods are useful and economical food. A few years ago Mr. Wilson retired from the business with a handsome competence.

The second firm that entered into the business of packing compressed meats was that of Libby, McNeill & Libby, who began the business in January, 1875, having arranged with the Wilson Packing Company for joint rights and privileges under the patents issued to them by the United States Patent Office, and believed to fully cover and protect all the processes employed in canning compressed meats. Next in the order of those who entered the field was a corporation styled the Saint Louis Beef-Canning Company. They began in Saint Louis, packing meats by the processes described by the letters patent issued to Wilson Packing Company, and to Libby, McNeill & Libby. Suits were immediately begun by the last-named two firms to restrain the Saint Louis Beef-Canning Company and others from using those processes in violation of the patents referred to above. The final result of these suits was a decision by the Supreme Court of the United States that the patents in question were invalid.

During the ten years that have passed since the canning of compressed beef began in Chicago the number of cases packed by processes followed in the business here exceed by several millions the total output of meats preserved by all other methods during the whole of the last three-quarters of a century. That the consumer has been benefited to a considerable extent by the change in methods of packing will be apparent to all who have seen goods packed by the old system, and have compared it with the firm, solid contents of cans filled under the new processes. The saving in cost of the amount of metal and of labor required for putting up meats by this method, and of transportation of that metal and the large quantity contained by the cans, is in the aggregate very large. In the business of a single Chicago firm 35,775,663 cans of beef have been put up. These contained, respectively, 1, 2, 4, 6, and 14 pounds each. Of these sizes the greatest number of cans sold contained 2 pounds. It may be assumed that they averaged 6 pounds each in weight, making a total of 214,654,000 pounds of compact meat, containing no bone and little, if any, moisture. If all this meat had been put up in the old way, accompanied by from 25 to 35 per cent. of moisture, the consumer would have been called upon to pay at least for the freight upon from 53,663,500 to 75,130,000 pounds of water. But the greatest gain to the consumers was in the improved flavor of the meats preserved by the new process.

The firms which entered the business from time to time after 1875, and which have retired therefrom, are mentioned below. Some were unsuccessful, and others retired for reasons unknown to the writer. A large number commanded ample capital, and did not, therefore, discontinue the business from any lack of means: American Meat-Canning Company, Saint Louis, Mo.; The Denver Packing Company, Denver, Colo.; Omaha Packing Company, Omaha, Nebr.; Kingan & Co., Indianapolis, Ind.; Hannibal Meat Company, Hannibal, Mo.; Slavins, Obern & Co., Kansas City, Mo.; Boston Beef-Packing Company, Fulton, Tex.; Chicago Packing and Provision Company, Chicago, Ill.; Chicago Meat Company, Chicago, Ill.; Ashwell & Co., Chicago, Ill.; Anglo-American Packing and Provision Company, Chicago, Ill.; James Turner, Chicago, Ill.

The firms now in the business in America, mentioned in the order in which they began, are as below: Libby, McNeill & Libby, Chicago, Ill.; Plankinton & Armour, Milwaukee, Wis.; Plankinton & Armours, Kansas City, Mo.; Lees, Hendricks & Co., Chicago, Ill.; Armour Canning Company, Chicago, Ill.; Fairbank Canning Company, Chicago, Ill.

From the beginning of the operations of the firm first in the list mentioned above, up to December 18, 1884, they had slaughtered 1,087,216 cattle, and had packed 35,775,663 cans of beef, which were distributed about as follows: To Great Britain, five-tenths; to Germany and all other foreign countries, two-tenths; the remaining three-tenths being consumed in the United States and Canada. Of that part of the supply of meats taken for domestic consumption in the United States, nine-tenths have been used in the homes of the people. Next in the order of the amount used come the Army and the Navy; then the merchant marine; third, the lumbering business; fourth, the mining camps, especially in the West; and fifth, railroad construction.

In a general way the cattle used in the canning business may be described as medium in grade. Canning companies buy some cattle of heavy weights and fine quality, but they make their greatest drafts upon the supply of Texan and Western cattle, which seem to be admirably adapted to the uses of the canning business. The supply of native cattle of medium grade is, during the summer months, comparatively light. Such cattle cost little or nothing while upon pasture, and gain flesh; therefore farmers prefer to keep them while pasturage lasts. When autumn comes such cattle are sent to market in large numbers, being then in fairly fat condition. During the summer months Texas and Western cattle reach Chicago, and supply the canners and packers with the bulk of the stock they use between June and December. It will be seen that in the absence of a full supply of other stock it is a matter of great importance to this business that ample receipts of cattle from the plains should appear in market during that season. The entire supply from other sources of stock suitable for canning would be, in the absence of Texas and Western cattle, too small to meet the wants of the market. Prices of beef would advance to almost prohibitory figures should any cause, such as a quarantine, for example, prevent the coming of the usual supply during the summer months. It has been held that, should such a restriction be imposed, the market would be glutted with cattle immediately upon the raising of the quarantine, and extremely depressed prices would result. This, it is thought by many, would offset the rise in prices resulting from a scarcity in summer. In the opinion of those who have been long engaged in the cattle business, this theory, while seemingly correct, would not be sustained by the results, as the demands of the several canning and slaughtering companies and others interested in the beef trade are so large that, however heavy the receipts of cattle might be for the time being, prices would be kept as high as though the receipts had been, as now, substantially regular throughout the entire year. It is possible that the general cattle traffic would adjust itself to such changed conditions and maintain an even supply; but serious disturbance of the trade would first occur, affecting a vast number, if not all, of the consumers of beef in this country. Undoubtedly a fear that such quarantine would be imposed and such disturbance result was one of the chief causes of opposition offered by dealers and others interested in these branches of the cattle trade of the West to the passage of the bill that created the Bureau of Animal Industry. It is now becoming understood by many of those most interested that such quarantine regulations as are required to prevent the spreading of the germs of Texas, or splenic, fever among the herds of the West and North might be rigidly enforced without interfering with the movement to market of beeves from the Southwest. Such cattle might be, as very large numbers are now, brought directly from Texas to Chicago by railroad. They would be unloaded at suitable places on the way, fed, watered, and rested, and reshipped without leaving the stock-yards. Protection would thus be afforded from so-called Texas fever to all cattle such as might be placed in cars, pens, or other places recently occupied by animals bearing the germs of that malady.

The English Government orders from Chicago houses large quantities of beef in cans for use in the army and navy of that country. France has several times offered to buy largely from a Chicago house beef preserved in cans; but those offers have been declined, because the French Government required that each can should be made to hold a kilogram, should be of a shape different from that in common use by the canning companies, and should be subject to inspection and rejection or approval upon arrival in France. A Chicago firm offered to furnish any required quantity of the goods in any desired form and size, upon condition that the French Government should station in Chicago an inspector or inspectors, to whom every process of the work, from the purchase of the cattle to the final sealing of the cans and their shipment, should be at all times open for examination, and who should day by day accept or reject the goods put up each day.

It may not be amiss to add here that no part of the live-stock business of the West is concealed from examination by any person who may wish to investigate. All animals offered for sale are inspected, especially in cases where there are reasons for thinking that they are not fit for food for human beings, by officers of the city health department. Slaughter-houses are open at all business hours, and admittance can be easily gained. Even to rooms where the several processes of canning are carried on, visitors are admitted upon application at the offices of the establishments.

In the year 1878, the two houses then engaged in putting up compressed cooked meats conceived the idea of supplying with choice cuts of beef the hotels, restaurants, and retail shops, which could not profitably use the beef from all parts of the carcass. Almost immediately the business grew to large proportions, and is now an important adjunct of the canning industry. The advantages the plan offered to all concerned were so plainly apparent and so great that but little if any opposition has been met to introducing such selected cuts. They now go to nearly every part of the country where the necessary facilities for transportation and for preserving the meat in its fresh state can be secured. Under this system a dealer in meats in the midst of a cattle growing district, as in Iowa, for example, may send his order for fresh beef to Chicago in the afternoon of one day, and early the next day receive the supply ordered. Having thus practically an unlimited number of carcasses from which to select, the buyer may confidently order any quantity of any desired part of the carcass, and be almost sure to receive, within twenty hours, all he ordered. Thus it is quite possible that the farmer who fattens a bullock, and one day sends it to Chicago, may, two or three days later, see upon his own table choice cuts of beef from that bullock, sent two hundred miles or more to be slaughtered and distributed. Shipments of such fresh cuts are directed to all parts of the country, while the other portions find their way in barrels, or in the form of canned compressed cooked meat, to the Eastern States, to nearly every country of Europe, and to all lands and seas to which the troops or the ships of Great Britain and of Germany are sent.

THE DRESSED-BEEF TRAFFIC.

The business of shipping dressed beef in refrigerator cars is of quite recent origin, but its growth has been enormous during the few years that have passed since its conception. From a very small beginning made by Mr. G. F. Swift, and perfected by him, his trade alone has increased from a single car-load to thousands of carcasses weekly. Mention has previously been made of the fact that the shipments of dressed beef from Kansas City was begun in the autumn of 1875 by Nofsinger & Co., of that city. The first shipment by that firm consisted of the carcasses of 103 head of cattle, two car-loads of which were sent through to Philadelphia and one car to Boston. The rate of freight to Philadelphia was

65 cents per 100 pounds; 75 cents per 100 pounds was charged for freight to Boston. On this lot of 103 cattle there was a profit of over $800. From 1875 to 1879 Nofsinger & Co. shipped dressed beef to Chicago, Saint Louis, Baltimore, Philadelphia, Boston, and other Eastern cities, with varied results. The railroads advanced their rates from time to time until the firm mentioned were forced to stop their shipments of dressed beef. During the time this firm were engaged in the business there were many difficulties in the way of carrying it on successfully. The supply of refrigerator cars was too small to answer the demands of the traffic, and many of those furnished were defective. It is said that railroad officials were unfriendly to the business, and that this was the cause of the continued advance in rates which left no profit for the shippers. It is also said that there was at times neglect on the part of the railroad employés in not supplying the cars with ice while in transit. For that reason beef would frequently reach its destination in bad condition. The delays in transit were often disastrous. The appliances in many of the markets where the beef was sold were crude and insufficient so that the meat got into bad order before it could be sold. Notwithstanding all obstacles put in its way, it is believed by those who have much experience that the shipping of dressed beef from Western packing points to the East is profitable to the shipper and beneficial to the consumer, and that in time capital, guided by intelligence, will overcome all difficulties which have thus far been in the way of the attainment of the highest possible results.

The number of firms engaged in the shipment of dressed beef is large and increasing, and it is evident that the old method of shipping live cattle in open cars from Chicago will be in time wholly superseded by the transportation of their carcasses in refrigerators, thus reducing the cost of beef greatly to the consumers, while the growers of cattle will receive more for their stock than they can under the present system, unless rates charged for the transportation of dressed beef shall be kept, as they now are, so high as to make the cost of the dressed beef as high as it would be if from cattle carried alive.

The following table shows, in tons, the quantity of dressed beef sent from Chicago to all points east of that city, over the New York Central and Saint Louis, the Chicago and Atlantic, the Chicago and Grand Trunk, the Michigan Central, the Lake Shore and Michigan Southern, the Pittsburgh, Fort Wayne and Chicago, the Chicago, Saint Louis and Pacific, and the Baltimore and Ohio Railroads, during the years 1881, 1882, 1883, and the first ten months of 1884. The table also shows the relative importance, in volume, of the dressed-beef traffic as compared with that in living cattle, swine, sheep, and horses and mules:

Years.	Dressed beef.	Cattle.	Swine.	Sheep.	Horses and mules.	Totals.
1881	33, 685	515, 502	156, 271	11, 938	3, 526	720, 923
1882	66, 075	492, 250	198, 287	15, 087	3, 707	775, 406
1883	154, 187	512, 823	158, 557	16, 269	5, 956	847, 793
1884*	151, 530	344, 371	138, 871	12, 504	8, 881	658, 157
Totals	405, 477	1, 864, 946	651, 986	55, 798	22, 071	3, 000, 278

*For ten months ended with October 31, 1884.

The table which follows shows the number of tons of dressed beef and live stock received, during the period mentioned above, in New York City, Philadelphia, Baltimore, Boston, and the New England States:

Years.	Dressed beef.	Cattle.	Swine.	Sheep.	Horses and mules.	Totals.
1881	75,259	658,385	317,810	18,101	8,261	1,077,266
1882	93,108	552,781	300,280	106,153	19,755	1,072,077
1883	145,871	604,149	345,962	147,617	28,962	1,272,561
1884*	141,535	428,650	270,623	119,245	27,904	987,957
Totals	445,773	2,243,915	1,234,175	391,116	84,882	4,409,861

* For the ten months ended with October 31, 1884.

A summary of the above two tables shows that the several sources from which came the total number of tons of dressed beef and of live stock received in the New England States, and in the cities of Boston, New York, and Philadelphia, during the period that began with the year 1881 and ended with October 31, 1884, contributed respectively as shown below:

From—	Dressed beef.	Cattle.	Swine.	Sheep.	Horses and mules.	Total.
Chicago	405,477	1,864,946	651,986	55,798	22,071	3,000,278
All other sources	50,296	378,699	582,189	335,318	662,811	1,409,583
Totals	455,773	2,243,915	1,234,175	391,116	84,882	4,409,861
Percentages, Chicago	88.97	83.11	52.83	14.26	26.00	68.04
Percentages, all other	11.03	16.89	47.17	85.74	74.00	31.79

Comparatively few of the cattle received at Chicago show evidences of disease. In September, 1882, a number of articles appeared in the papers of Chicago, declaring that numbers of diseased animals were taken from the stock-yards and slaughtered for food for human beings. The articles were very graphic in their description of scenes in the slaughter-houses and stock-yards, and were copied widely in the Eastern States, as well as in Europe. Although it was explicitly stated that great care was used, by canners and by shippers of dressed beef, to prevent animals unfit for food being slaughtered for their uses, the effect of those articles was damaging to the interests mentioned. The principal packers and shippers of beef at once exerted themselves to assist the city health department of Chicago in its efforts to prevent the sale of any animal suffering from disease, or from injuries that made their flesh unfit to eat. The department mentioned has authority for this purpose over the entire area within a line 1 mile outside of the limits of the city. An inspector is stationed at each slaughter-house, and one is constantly on duty at the stock-yards during those hours when the gates of the yards can be opened for the passage of stock. Those officers scrutinize the animals offered for sale, especially those destined to be slaughtered in Chicago. If an animal exhibits indications of a disease making the flesh unsuitable for food, it is condemned, and goes direct to the establishments where its carcass is converted into fertilizers.

Inspectors are also stationed at the wholesale markets, where they carefully examine all meats offered for sale. When meat is condemned kerosene oil is poured over it. The odor of the oil makes it quite impossible to dispose of the flesh for food. All meats offered in the markets of Chicago undergo this inspection, as do all cattle offered for sale alive. Employés of the canning companies and of shippers of dressed beef report to their principals, or the inspectors, the presence in the markets of any diseased animal, and condemnation quickly follows. These facts seem to warrant the assertion made that the meat supply of Chicago is practically entirely wholesome. Self-interest leads the packers and the canners to use every available means for preventing even the shadow of suspicion resting upon the goods they have to sell; hence they become most efficient aids to the health department, which has given proof of efficiency in this branch of its work.

The extent of the appearance of disease in the live stock marketed in Chicago is indicated by the figures which follow, showing the number of cattle, hogs, and sheep, and of pounds of meat condemned in Chicago during the year 1884:

Cattle:		
Diseased		515
Crippled		301
Total condemned		816
Hogs:		
Having cholera		1,840
Crippled		138
Total condemned		1,978
Sheep, emaciated or crippled		2,050
Calves, prematurely born (slunk)		57
Meat condemned:		
At the Jackson street market	pounds	332,164
At the South Water street market	do	229,301
Total condemned within the city limits		561,465

Since the number of cattle received at Chicago in the year 1884 was 1,817,697, of hogs was 5,351,967, and of sheep was 801,630, it will be seen that, if it be assumed that all animals not fit for food were condemned, the percentage of such animals was: Of cattle, .000449; of swine, .00037; and of sheep, .0025.

Large herds of Texas cattle are driven through the streets of Chicago, or over the prairies adjoining. They are followed by milch cows upon which hundreds of households depend for a large part of the food they require, particularly for their children. These cows go each morning in summer to graze on the commons west and south of the city. Cropping here and there a blade of grass as the herd boy drives them along, they get the germs of the fatal Texas fever, and in a few days the family misses its dumb friend and provider. Hundreds of milch cows have been lost from this cause in Chicago. It has often been suggested by those interested in the cattle traffic that a portion of the stock-yards should be set aside for the exclusive use of Texas cattle. In the portion of the yards assigned to that branch of the business no native cattle should be allowed during those months when there would be no frost to destroy the germs of the fever. It has also been urged that there should be in the stock-yards and under the supervision of State officers, a slaughter-house, to which all crippled, bruised, or diseased animals should be sent, there to be killed for the benefit of the owner. Under such a system as that suggested, it is claimed the owner would receive for his stock all it was justly worth, since if upon inspection the carcass or any part of it should be found fit for human food it could be sold for that purpose. If not fit for such use it would be condemned to the rendering tank, and thus be prevented from entering into consumption as food.

There is no doubt in the minds of those acquainted with the cattle traffic that some animals suffering from tuberculosis are sold in the cattle markets of the West, and their flesh is eaten by human beings. There is no data showing how large a percentage of the entire receipts are thus affected. It is possible that a careful investigation at the slaughter-houses would result in obtaining statistics that would show this matter clearly, but no systematic effort has been made to obtain such data. Occasionally physicians and others have made brief examinations, but these were disconnected, and resulted in obtaining no information tending to show how many cattle exhibit lesions of this malady.

There is every reason for believing that no case of contagious pleuro-pnemonia or of foot-and-mouth disease has ever appeared in any of the stock-yards of the West. It is evident, however, that there is nothing in the methods of the trade which would prevent the very rapid spread of any contagious or infectious disorder, should such once appear in any of the stock-yards along the railroad lines over which the cattle traffic is carried on. No means for disinfection have ever been used here in cars, pens, or yards in which cattle are confined, and it is clear that the use of such disinfectants would involve a considerable outlay of labor and of money. There is no inspection of any of the material of the trade after its use by one lot of cattle before another follows; so that in case of the use of cars or yards by animals suffering from contagious or infectious disease others may follow immediately after, to become infected and extend the ravages of the malady.

Respectfully submitted.

EDWARD W. PERRY.

CHICAGO, ILL., *December*, 1884.

CATTLE INTERESTS OF THE WEST.

REPORT OF J. H. FULLINWIDER, OF KANSAS, INSPECTOR OF THE BUREAU OF ANIMAL INDUSTRY.

Hon. George B. Loring,
Commissioner of Agriculture:

Sir: I have the honor to submit the following report of the work done by me since my appointment as Inspector of the Bureau of Animal Industry, in August last.

The country west of the Mississippi River was assigned to me as the district to which I was to give my immediate attention, and within which my investigations were to be confined. In a district so vast, and with but three months in which to work, the labor to be performed has necessarily, in many directions, been but partially completed. However, in some lines of investigation I have been enabled to proceed far enough to reach reliable conclusions.

CATTLE INTERESTS IN THE WEST.

The cattle of the United States are pretty nearly evenly divided between those States east of the Mississippi River and those lying west of that stream, *i. e.*, 23,000,000 head, or a little over, on either side. There is, however, this great difference; in the eastern portion of the country, a large proportion of the cattle are kept for dairy purposes, while in the western portion cattle are raised almost exclusively for beef, the important exceptions being in the States of Minnesota and Iowa, in both of which dairying is carried on quite extensively. The beef raising or cattle industry of the West is in the hands of two great classes, the ranch-men and the farmers. These two classes carry on the business upon such widely different principles that often a statement in regard to one class will be found entirely at variance with the facts in connection with the other class. For example, the cost of raising beef on the ranch is about $2 per cwt. weight, while on the farm (corn-fed) the cost is nearly or quite $4 per cwt. This difference must be borne in mind when dealing with the cattle interests of the West taken as a whole.

The greater portion of the beef cattle of this portion of the country are what are termed "native" or common stock, this being especially true of Texas, the greatest cattle-growing State in the Union. Many breeders are, however, exerting themselves to raise the standard of the "range" cattle of this section of the country by the introduction of thorough-bred bulls. This effort is being crowned with success, unless it be in Texas and other portions subject to Texas fever, it being found difficult to introduce blooded stock that will withstand this scourge.

Heretofore there has been an opportunity for the small owner, with his few hundred head, to enjoy range privileges, as well as his more wealthy brother with his thousands of cattle; but at the present time there appears to be an inclination on the part of the large owners to crowd out the small men—a disposition among the big fish to eat the little ones. One by one the small bands of cattle are disappearing and the ranges are being taken possession of by the large cattle companies and "pools." The farmer, too, is wide-awake and seems to fully appreciate the value, to him, of blooded or at least of graded cattle, and their superiority, from a paying point of view, over the native stock. While this is a matter of vast importance to both ranchmen and farmers, it is particularly so in the case of the latter. The difficulty in getting blooded cattle that can stand the "rustling" life of ranch stock may interfere to some extent with this enterprise on the plains, but with the farmer everything is in favor of the blooded animal. From the limited space at his command it is important that the farmer get the greatest amount of beef attainable from the smallest number of cattle, and here is where the thorough-bred and "graded" cattle come to his assistance.

It is a fact, established beyond successful denial, that a thorough-bred or graded Shorthorn or Hereford steer will cost no more to raise than a native, and that at the same age he will weigh more, and command a more ready sale at a better price.

FAVORITE BEEF BREEDS.

In selecting the strain of blood with which to improve his herd, which of the many now in the country shall the breeder choose, his object being beef, and beef alone? This is a question about which there is a good deal of difference of opinion, with the weight of testimony in favor of the Shorthorns (Durham) and Herefords. Out of one hundred and twenty-four correspondents who expressed an opinion on this question, sixty-seven favored the Shorthorn (Durham) cattle, and about one-half that number preferred the Herefords, the Polled Angus and Galloways coming in for a small vote, each being preferred in the order named. One very intelligent breeder prefers a cross of Polled Angus and Shorthorn, as one combining the large frame of the Angus with the rounding, finely "filling out" qualities of the Shorthorn. In Texas the crossing of Texas cows with Hereford or Shorthorn bulls is recommended.

COST OF BEEF PRODUCTION.

The cost of raising a three-year-old steer, that will weigh from 1,000 to 1,200 pounds, varies greatly in different parts of the district we have under consideration, owing to the differences of climate and manner of handling; and again it varies a great deal in the same State or Territory, depending to a large extent upon how much grain is fed, capital invested in land, &c.

Below I give, as near as could be ascertained, the cost per 100 weight to the producer of a three-year-old steer, weighing from 1,000 to 1,200 pounds. It must be understood that the less amount generally refers to grass-fed cattle, and that the higher figures refer to grain-fed stock:

Kansas:		
Range steer	per cwt..	$0 50 to $2 00
Range steer, beef fat	do....	3 00
Farm steer, beef fat	do....	4 00
Nebraska:		
Range steer	do....	1 00 to 2 87
Range steer, beef fat	do....	3 00
Farm steer, beef fat	do....	4 00

Minnesota:		
Farm steers (raising)	per cent..	$1 00 to $2 90
Farm steers, beef fat	do....	3 00
Missouri:		
Grass fed	do....	40
Corn fed	do....	3 00
Montana; All grass fed	do....	30 to 1 00
Dakota: Grass and corn fed	do....	1 25 to 3 50
Texas: Grass fed	do....	65 to 66
Idaho: Grass fed	do....	1 50

As indicated above, these figures are for steers three years old. This age was taken because it is the age at which a large majority of cattle are turned to market. Our correspondents reported from fifty different localities that three years was the age at which cattle are turned to market; from thirty-three localities, that four years was the average age of a beef steer; and from twenty-eight localities, that steers were turned to market at two years of age. An interesting circumstance in this connection is that cattle are turned to market at two years old in those localities where the greatest number of Shorthorn or Hereford grade cattle are found; and just in proportion as the number of thoroughbred or graded animals decreases, the age of turning to market increases; and hence it is of importance to the breeder of cattle for beef to introduce into his herds pure blood of some good beef-producing strain.

LOSS IN FLESH AND DEATH RATE FOR WANT OF SHELTER.

The cattle west of the Mississippi River are in excellent condition at this season of the year (November), most localities reporting the condition as "unusually fine," "fat," "fit for beef," &c., and this is the condition in which cattle on the ranges usually start into the winter; but as, in a vast majority of cases, they have no shelter other than that afforded by timber or bluffs, they come out in the spring having suffered a loss in flesh of from 1 to 15 per cent. In addition to this, the death rate during the winter frequently reaches 10 per cent. Nearly all of this enormous loss might be obviated by an outlay that is trifling compared with the amount that would be saved thereby. By the erection of proper barns and sheds the producer would bring his cattle through the winter with but very slight loss. Straw sheds, with an eastern exposure, will be found as good as anything that can be devised. To show in a striking manner the beneficial influence of some kind of artificial shelter, I give below in tabular form reports upon this subject that were received by me from several States and Territories:

MINNESOTA.

County.	Fall condition.	Kind of shelter.	Loss of flesh.	Death rate.
Meeker	Good	Barns and stables	None	
Wadena	Good	Stabling		None.
Olmsted	Fat	Sheds and stables	Gain. 2 per cent	None.
Waseca	Fat	Poor	15 per cent	
Watonwan	Good	Straw stacks	Grow thin	Very few.
Jackson	Good	Good stables	None	None.
Stearns	Good	Barns	None	None.

DAKOTA.

County.	Fall condition.	Kind of shelter.	Loss of flesh.	Death rate.
Grant	Beef order	Warm stables	None	None.
Hutchinson	Good	Sheds	Some loss	
Do	Good	Stables	Small gain	
Pembina	Fine	Stables	No loss	None.
Sanborn	Splendid	Poor		10 per cent.

NEBRASKA.

County.	Fall condition.	Kind of shelter.	Loss of flesh.	Death rate.
Cass	Good	Straw sheds	Gain	
Thayer	Good	Straw sheds	No loss	None.
Cherry	Good	Canopy of heaven		15 per cent.
Filmore	Good	Straw sheds	Loss 2 per cent	
Colfax	Good	Straw and lumber sheds.	None	Very little.
Harlan	Good	None		3 per cent.
Richardson	Good	Barns and sheds	Very slight	Small.
Buffalo	Good	Sheds	Very little	Light.
Platte	Fair	Open sheds	10 per cent	5 per cent.

MONTANA.

County.	Fall condition.	Kind of shelter.	Loss of flesh.	Death rate.
Meagher	Very good	Hills	10 to 15 per cent	
Dawson	Good	Bad lands	10 per cent	5 per cent.
Custer	Fat	Nature	5 per cent	Little.

TEXAS.

County.	Fall condition.	Kind of shelter.	Loss of flesh.	Death rate.
Galveston	Good	None	Become thin	10 to 15 per cent.
Collin	Thin	None		1 per cent.
Cooke	Good	Timber	10 per cent	5 per cent.
Johnson	Good	None		10 per cent.

KANSAS.

County.	Fall condition.	Kind of shelter.	Loss of flesh.	Death rate.
Marshall	Good	Hay sheds		Very light.
Ford	Splendid	None	Lose all winter	2 per cent.
Sheridan	Good	Natural	10 per cent	3 to 5 per cent.
Waubaunsee	Good	Timber	7 per cent	2 per cent.
Barton	Good	Not much	Considerable	5 per cent.
Davis	Good	Very little		2 per cent.
Edwards	Good	Straw or corral	10 per cent	5 per cent.
Barber	Good	None		3 to 8 per cent.
Pawnee	Good	Very little	15 per cent	

Compare Marshall County, Kansas, where hay-sheds are used, with Sheridan County, Kansas, where there is no shelter except such as is afforded by nature. Where the hay-sheds are used the loss is almost nothing; where the cattle have nothing but "natural" shelter the loss in flesh is 10 per cent., and the death rate is from 3 to 5 per cent. Follow the same comparison throughout the table, and no argument will be necessary to induce the ordinary farmer to shelter his cattle, be the mode ever so rude. As has been suggested above, nothing is better than a good straw shed.

SHIPMENT OF DRESSED BEEF.

Having fattened his cattle so that they are ready for beef, how is the stockman to send this beef to market? Shall it be as live stock or in the shape of dressed beef? Until quite recently the answer would unhesitatingly have been "as live stock." But to-day the project of slaughtering beeves at some railroad point near the cattle ranches of the West and shipping the dressed beef in refrigerator cars to the eastern markets is one receiving increased attention from cattle men, and is enthusiastically indorsed by most of them. It requires a large amount of capital to carry on this business successfully, but as it is one paying large profits, capital is easily obtained. A large concern is soon to be built in Omaha, with a capacity of 600 beeves per diem, and if strict quarantine is enforced against Texas cattle in the North the stock-raisers of that State will be compelled in self-defense to resort to abattoirs and refrigerator cars. This manner of shipping beef is of great economy and perfectly practicable, and will soon be one of the greatest savings of agricultural commerce, as it would distribute the supply through the year and not throw so much beef upon the market in the months of August, September, and October. There is undoubtedly an opportunity for the establishment of an exceedingly profitable business at several railroad points in the West, Northwest, and Southwest. These should be within easy driving distance from the large cattle-raising centers of the respective regions.

EXISTING DISEASES AND MODES OF PREVENTION.

When the vast extent of that portion of the United States lying west of the Mississippi River is taken into consideration, this section of the country may be said to be remarkarkbly free from diseases among its cattle; and yet when the individual cases of disease from the various States and Territories are taken in the aggregate they amount to an enormous loss.

TEXAS FEVER.

The most terrible disease known in the district under consideration, because the most fatal and the most difficult to control, is Texas or splenic fever. I shall not attempt to expand upon Dr. Salmon's very full and able discussion of this scourge, in Report of Contagious Diseases, issued by the Department in 1880, but shall content myself with the report of cases which have come under my immediate observation since the first of last August, and the discussion of the various modes suggested for the prevention of this disease.

Mr. Lake F. Janes started from Ohio with fifty head of fine cattle. At Decatur, Ill., they were unloaded for food and rest. There were at the time these cattle were in the Decatur yards some other cattle there *sick*, though it is not positively known that they were suffering with Texas fever. Seven days after he left Decatur with his cattle Mr. Janes noticed that some of his cattle were sick with what proved to be Texas fever. He lost ten head, and would probably have lost more but for the timely use of green corn and salts.

The Messrs. Robertson Brothers, of Ohio, brought twenty head of fine Shorthorn cattle to Kansas by the way of Chicago and Hannibal, Mo., at which latter place they were unloaded for feed and rest, except some extra fine bulls, which were left in the car. Shortly after arriving in this State thirteen of his *cows* were taken with Texas fever, and nine of them died. It is to be remarked in this case that the bulls, which were not unloaded at Hannibal, remained perfectly healthy, proving almost conclusively where this herd were exposed to this scourge.

In 1883 five hundred head of Arkansas cattle were driven into Harper County, Kansas. From this herd Texas fever was scattered over the whole county, one thousand head of cattle dying in that county alone. Many small cattle dealers were almost broken up, and farmers' herds were decimated. The man who had driven in the herd had his cattle taken from him, damages for the cattle lost and a fine of $300 assessed against him, and was himself lodged in jail.

Up to November 15, 1884, cattle were dying of Texas fever in Dodge City, Kans. One herd of six hundred had suffered a loss of one hundred and fifty head. In Lyon County fifty died out of a herd of three hundred, and Captain Adams, of Shawnee County, Kansas, lost one hundred and twenty out of two hundred. So wide-spread was the fear of this disease the past fall in Kansas that thousands of cattle were prematurely shipped to market, the owners preferring to market their stock in poor condition rather than take the risk of losing them by Texas fever.

During the summer of 1884 seven head of cattle died of what proved to be Texas fever at Rockville, Mo. Investigation showed that no Texas stock had ever been in the town, *except as they passed through on the cars en route* for market. Further investigation showed that all the cattle that died had *grazed along the railroad track*, where were found quantities of straw litter and manure which had evidently fallen from the cars.

In the spring of 1884 a herd of Texas cattle were driven through Cherry County, Nebr., on their way to Dakota. The result was that in that county alone 2,000 head of cattle died of Texas fever during the past season. This herd traveled all the way from Texas to Dakota, endangering thousands of cattle all along their route.

Instances might be multiplied to show the fearful ravages of this disease among the herds of the Northern States, and in almost every case it is traceable to Texas stock on their way to Montana or Dakota. Strict quarantine laws, rigidly enforced against all Southern cattle, except during the months when heavy frosts are experienced, is the method of prevention most strongly recommended by the cattle-men north of the danger line. So far as the Northern breeder alone is concerned this may be as good a method as any that can be devised. But the cattle-men of Texas and other southern portions of the country object that such a method necessarily entails great loss upon them. This problem should be so solved, if possible, as to protect both the cattle-men of the infected and non-infected regions. Two methods have been suggested to accomplish this object:

(1) The establishment of a national cattle trail, several miles in width, with clearly defined limits, and extending from the Red River of the South to the Red River of the North, along which trail Texas cattle may be driven at any season of the year. That when this trail is established Texas cattle be prohibited, by severe penalties, from passing along any other route. And, further, the law establishing the national cattle trail shall forbid the transportation of Texas cattle northward by railroad or otherwise, except during the months when severe frosts are experienced.

(2) It is claimed, and the case mentioned above at Rockville, Mo., seems to substantiate the claim, that Texas fever is communicated to healthy, native stock by litter, straw, and manure, which drops from cars on their way north with Texas cattle.

There can hardly be a doubt that this disease is communicated to cattle transported in cars previously used for the conveyance of Texas stock; or that cattle confined in the same pen with Texas cattle, or in pens where the latter have been confined since sharp frosts have fallen, are thereby given this disease. This being the case, it is demanded by the cattle-men of the non-infected regions that, if Texas beeves are to be allowed to go north during the summer months, the railroad companies be compelled to provide separate cars for the transportation of Texas stock; that these cars be plainly branded, and so constructed that no straw, litter, or matter of any kind can possibly drop from the car while *en route;* that separate pens be provided at the resting places, to be known as Texas pens; that only Texas stock be unloaded in these pens, and that it be made a misdemeanor to unload or, under any other circumstances, to place Texas cattle into any pens except those expressly provided for them. And lastly, that the railroad companies be compelled to destroy, by burning, all litter taken from cars used for transportation of Texas cattle immediately upon arriving at their destination, and to thoroughly disinfect these cars at once. The cases of Mr. Luke F. Janes, at Decatur, Ill.; of Messrs. Robertson Brothers, at Hannibal, Mo.; and of the Rockville, Mo., cattle, certainly afford ground for these demands, and I would most respectfully recommend that Congress be urged to put so much of the demands indicated above as may be deemed wise and practicable into the form of law. The possible, and not at all improbable, solution of this problem will be that Texas will ship her *beef* cattle to market *dressed* and in refrigerator cars, and that the cattle which are annually taken from that State to Montana and Dakota to fatten will be driven north along a national cattle trail.

BLACK-LEG.

It is quite astonishing to observe the general extent of this disease among the young cattle of the Northern United States. Reports from all the States and Territories have not been received, but Minnesota, Montana, Kansas, Nebraska, Idaho, and Dakota have all reported it as existing among the young cattle of their respective localities. All concur in the statement that the loss upon individual owners is small; and yet when the general prevalence of this disease is taken into consideration the total is no small matter. It attacks young cattle almost exclusively, and frequently has advanced so far before its presence is discovered as to be beyond successful treatment. It is claimed by a large number of our correspondents that by giving plenty of salt, saltpeter, and sulphur animals suffering with black-leg, that from 25 to 30 per cent. can be saved. Among the remedies used for the treatment of this disease the use of salt, saltpeter, and copperas has been found beneficial, as has also a mixture of salt and air-slacked lime. Others recommend salt and saltpeter without either sulphur or copperas. A mixture of pitch-tar and lard will be found beneficial in many cases. Roweling is a method resorted to by many, and the vaccination of young stock is followed to some extent. I think I am justified in saying that the timely use of salt, saltpeter, and sulphur is the most effectual mode of treating this disease at present known to our stockmen.

THE GENERAL CONDITIONS OF HEALTH AND METHOD FOR PREVENTING DISEASES.

This subject has been quite freely discussed by my correspondents, and with a remarkable unanimity. The three conditions of pure water, wholesome food, and good shelter are the main reliance of every intelligent stock-raiser who is laboring to improve the condition of his animals. There is a mistaken idea among many farmers that any water is good enough for cattle to drink, and they therefore allow their horned cattle to drink, during the summer months, from stagnant pools of muddy water; in many cases the wallowing holes for hogs and the drinking places for cattle are one and the same; and then when his animals begin to suffer, and mayhap die, from one or another of the diseases which are but the natural results of drinking such impure water, he reports that his cattle are "dying from some mysterious cause," to him unknown. It behooves all stock men to see that a plentiful supply of pure water is furnished to his cattle, and to keep a vigilant watch upon this supply to see that it does not in any way become tainted.

Unwholesome and damaged food is often given to cattle, "*to save it.*" Never was there a more mistaken idea. The grain, it is true, is eaten, but the cattle are not benefited by it, and are almost sure to be injured; and if nothing more is done, the animals are kept at a standstill when they would otherwise be improving.

Somewhat has already been said in regard to good shelter as preventing loss of flesh and diminishing the death rate. This is one of the three things insisted upon by all intelligent stockmen. It need not be a costly barn. In all except the extreme northern section of the country straw sheds open to the south or east will be found as effectual as any kind of shelter that can be provided. This shed usually need cost nothing more than the two or three days' labor expended in hauling the poles and straw. A straw shed with straw properly stacked around it will more than pay for its cost in the feed the straw will be to the stock and the manure thus obtained.

FUTURE OF THE CATTLE INDUSTRY.

There are west of the Mississippi River 23,077,611 cattle, an increase since 1880 of 7,537,132, or over 1,750,000 head per annum. Nothing more is needed to demonstrate the vastness of this enterprise. The business of raising cattle is in the hands of two great classes, *i. e.*, the men who raise cattle on and sell them from the range, and the farmers who each year turn from one to five hundred head of beeves to market. The interests of these two great classes are closely united, and they are of mutual benefit to each other. The range man is able in the fall of the year to turn over to the farmer such of his herd as are not at that time fit for market, but would make fine beeves by the next spring if properly fed during the winter. The range man has neither the feed nor the facilities for this part of the business, and finds it more profitable to sell a steer at two years past than to hold him till three years past. On the other hand, the farmer of our Western States and Territories will seek in vain for a method of marketing his corn, straw, and hay that will prove as profitable as the one which sends them to market in the shape of corn-fed beef. Hogs are always, or nearly always, fed in connection with cattle, thus reducing waste to the minimum. In this connection the question of the superiority of thoroughbred or grade cattle over natives again comes up.

If a farmer can handle but one hundred head of cattle each season, it behooves him to obtain cattle that will give him the greatest increase in size and weight in repayment for the grain fed. It costs no more to care for and feed a grade Shorthorn or Hereford steer than it does a native, and yet it is established beyond doubt that the native will not make him anything like as large a return for his outlay as either of the grades above mentioned would do; and when he has his beeves ready for market the man who offers the grade can always command an advantage over the native scrub. The future of this industry will be the raising of cattle on the ranches to be fattened for market by the farmers of our large corn-growing States. These beeves will be slaughtered at the abattoirs in the West and shipped to the East as dressed beef.

LEASING PUBLIC DOMAIN, NATIONAL TRAIL, ETC.

The large cattle companies and wealthy stockmen of the West say that the western country is settling up so fast and the number of cattle increasing so rapidly that their interests are becoming insecure, and that in a few years they will be crowded out. They are, therefore, to ask the Government to lease to them the public domain at one cent per acre, and urge as one reason why the Government should do so, that $12,500,000 would thus be annually added to the public revenue. This, at one cent per acre, would give 1,250,000,000 acres of land which they would control. This very claim of the enormous revenue that would be paid to the Government shows the untruthfulness of the statement that the western ranges are overcrowded with cattle. Our correspondents from all over the West give less than 10 acres on an average as the number required for the support of one head of stock. To avoid doing these gentlemen injustice, let us put the number of acres required to support one head at 12.5. Now, multiply the 23,000,000 cattle west of the Mississippi River by 12.5 and we have 287,500,000 acres as the number required to support all the cattle of this region, leaving nearly 1,000,000,000 acres that are not yet stocked. This vast amount of land is the prize for which these wealthy cattle companies, largely controlled by foreign capital, are striving. While the cattle interests should be fostered, and every wise and honest effort made by them assisted, such schemes as that of leasing the public domain should be discountenanced.

The question of a national cattle trail is another one that should not be passed upon without the closest scrutiny. It is a remarkable feature in the discussion of this question that while many are ready to advocate a trail, they all object to having it pass anywhere in their own immediate vicinity. This would seem to indicate that while the trail is acknowledged to be a good thing for Texas, it is considered a bad thing for the country through which it may pass; and it would seem but just that no community should be forced by Congress to practically retire from the cattle industry simply that Texas cattle-growers may render their business more profitable.

As has been heretofore remarked in this report, a national cattle trail may be found necessary; but it should be established only after thorough investigation, and not then unless such investigation conclusively shows that it can be conducted without any great injury, either present or prospective, to the section of country through which it may pass.

Respectfully submitted.

J. H. FULLINWIDER,
Inspector, Bureau of Animal Industry.

EL DORADO, KANS., *December*, 1884.

SYNGAMUS TRACHEALIS.

EXPLANATION OF PLATES.

PLATE I, FIG. 1.—The trachea of an adult pheasant, whose death was caused by the gapes, slit open longitudinally, and showing, in its interior and attached to the mucous membrane, about thirty pairs of syngames in various stages of development (natural size).

FIG. 2.—A pair of syngames, attached with the mouth of the male and that of the female (enlarged 4 diameters).

FIG. 3.—A pair of syngames enlarged 10 diameters; A, male; B, female; showing the intestinal canal, the œsophagus, and the buccal capsule. In the female B may be seen, in addition, the uterus and its horns filled with ova and the ovarian tube coiled around the uterus and the intestine. In the male A the testicle is seen coiled about the digestive tube.

FIG. 4.—Mouth of a female syngame; A, seen from its face; B, from the side (enlarged 40 diameters).

FIG. 5.—Portion of the neck of a female (enlarged 25 diameters), showing at *a a* the cuticle finely striated; at *b* the subcutaneous, fusiform, muscular fibers; at *c* the œsophagus; at *d* a salivary gland; and at *e* the anterior extremity of the intestine into which the œsophagus opens, and which is seen lined with hepatic cells.

PLATE II, FIG. 6.—Reproductive organs of the female (enlarged 8 diameters); *a*, uterus; *b b*, uterine horns; *c c*, oviducts or Fallopian tubes; *d d*, ovaries.

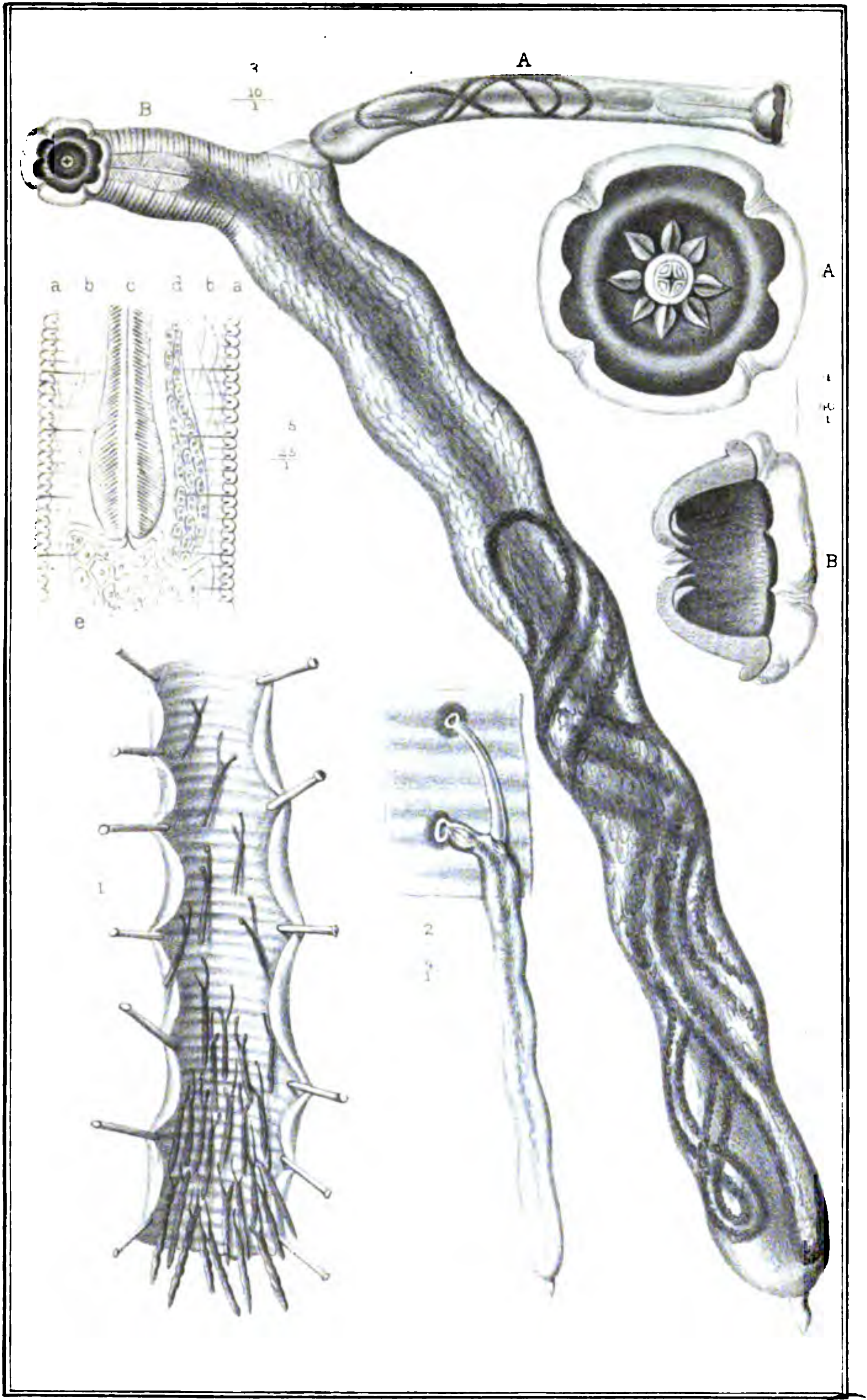
FIG. 7.—Reproductive organs of male (enlarged 20 diameters); *a*, spicules; *b*, spermatic canal; *c*, vesicula seminalis; *d*, testes.

FIG. 8.—Ova in different stages of development (enlarged 260 diameters). A, vitellus, segmented and muriform; B, ovum with granular vitellus, becoming constricted at its middle, the embryo developing laterally; C, ovum with embryo fully developed, folded like the figure 8; D, ovum with the valves at the extremities detached, and the embryo emerging.

FIG. 9.—Embryo directly after leaving the egg (enlarged 260 diameters).

FIG. 10.—Embryo somewhat older, undergoing the first molt (same enlargement).

FIG. 11.—Nymph (enlarged 100 diameters); *a*, rudiment of the genital organ.



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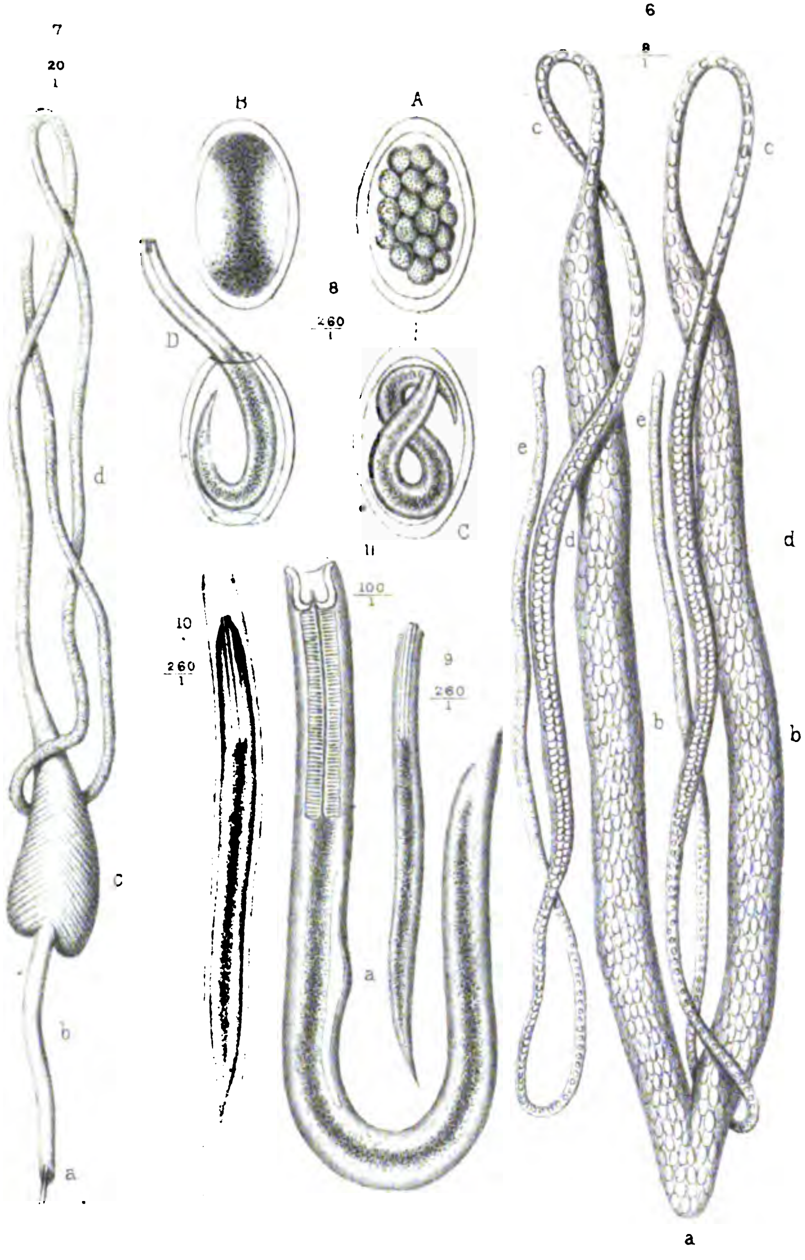
THE GAPE-WORM OF FOWLS

Syngamus trachealis (v. Siebold)
Sclerostoma syngamus (Diesing)

UNIV. OF MICH.



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THE GAPE-WORM OF FOWLS

Syngamus trachealis (v. Siebold)
Sclerostoma syngamus (Diesing)

THE GAPE DISEASE OF FOWLS, AND THE PARASITE BY WHICH IT IS CAUSED.

MEMOIR ON A VERMINOUS EPIZOOTIC DISEASE OF THE PHEASANTRIES* AND ON THE PARASITE WHICH CAUSES IT, THE SYNGAMUS TRACHEALIS (SIEB.), SCLEROSTOMA SYNGAMUS (DIES.), BY M. P. MEGNIN, LAUREATE OF THE INSTITUTE (ACADEMIE DES SCIENCES), MEMBER OF THE SOCIÉTÉ DE BIOLOGIE, HONORARY ASSOCIATE OF THE ROYAL VETERINARY COLLEGE OF LONDON, ETC.

[Translated by Dr. THEOBALD SMITH.]

For several years past the pheasantries of the hunting forests of France have been ravaged by a most destructive malady, which has killed the fowls by the hundreds and even thousands. The cause is a parasite, a so-called red worm, which develops in the trachea of birds and finally suffocates them. Particularly the young subjects, from six weeks to three months of age, are apt to be the victims, although adults by no means are always spared. The chief symptoms of this affection are a suppressed or aborted cough and a characteristic gaping, whence is derived the English name "gapes." It appears to have been observed long ago in England and America, whilst with us it has not yet been studied, a fact which seems to indicate that it has been introduced from England, and that we owe its introduction to commerce by which the hunting grounds have been restocked.

I investigated this disease on the site of its activity in the inclosures of the forest of Fontainebleau in 1878 and 1879. I received many cadavers killed by the red worm from different localities of central and northern France; from the poultry-yard of Baron Rothschild, at Rambouillet, where the daily losses amounted to 1,200; from M. de Janzé, of Gournay; from the duchess de la Rochefoucault, at Montmirail; from the inclosures at Chauteau-neuf, and from various localities of Loiret and de l'Indre. Finally a dispatch, in October, 1880, informed me that the epidemic had appeared in the royal pheasantries at Turin, and was threatening to do much mischief.

This disease is not at present raging on the continent only. For ten years it has been the cause of severe losses in England. Dr. Crisp estimates that the red worm destroys annually half a million chickens, excluding pheasants and partridges, so that he says it would be of truly national importance to find the means of preventing the invasion of this red worm or of destroying it.[†] Furthermore, the following statement is taken from the report of the meeting of the London Entomological Society, October 1, 1879:

> The president announced that Lord Walsingham, in conjunction with other gentlemen, had placed at the disposal of the council the sum of £100 to be awarded in two prizes of £50 each for the following subjects:
>
> 1. The best and most complete life history of *Sclerostoma syngamus*, supposed to produce the so-called gapes in poultry, game, and other birds.

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[*] This monograph, finished November, 1880, has reference to the epidemics in the pheasantries of France.

[†] *Path. Society of London*, October 15, 1872, and *Med. Times*, 1872, p. 474.

2. The best and most complete life history of *Strongylus pergracilis* (Cob.), supposed to produce the grouse disease.

No life history would be considered satisfactory unless the different stages of development were observed and recorded; the competition was open to naturalists of all nationalities. Essays in English, German, or French were to be sent to the secretary of the society on or before October 15, 1882.

Although birds only are concerned in this matter, it is obvious that the economic interest involved in a solution of the questions concerning the gapes is sufficiently great. The scientific interest is no less so, because there are to be determined not only the zoological position of the worm under consideration, and its rôle in the terrible disease which destroys the gallinaceans, both domestic and wild, but also its mode of reproduction, a point hitherto entirely unknown.

This is the subject of the present memoir, a memoir in which I believe I have cleared up all the pending questions upon the zoological position of the red worm, on its anatomy and physiology, on its rôle as a cause of the gapes, finally on its embryogeny and metamorphosis, and consequently upon its mode of propagation, and upon the best means of preventing its multiplication and arresting its ravages.

HISTORICAL.

The first mention of this disease was made by Dr. Wiesenthal, who observed it in 1799, at Baltimore, Md., among hens and turkeys.[*] In 1806, 1807, and 1809, Géorges Montagu[†] saw this epizootic among chickens in England. He believed that of all the birds of the poultry yard only the hen could be its victim, because he observed that the turkeys and ducks living with the infested hens were not attacked. He observed the same malady in young pheasants at a time when they assume the livery which distinguishes the two sexes, and in partridges whether the locality was elevated or low and humid.

Both Wiesenthal and Montagu recognized that this disease was caused by worms occupying the trachea and extending occasionally to the pharynx, but never as far as the lungs. They found as many as twenty attached to the mucous membrane, which, together with the lungs, was in an inflamed condition. These entozoa, acting finally as an obstacle to the passage of air, produced death by asphyxia.

Wiesenthal did not occupy himself with the specific determination of the worm, but Montagu regarded it as a distome, a fasciola (fluke) of a particular kind, having a round cylindrical body with two sucking disks, borne on two peduncles of unequal length.

Rudolphi[‡] and the authors of his time continued to regard the cause of the gapes in the gallinaceans as a distome, and included it in the species *Distoma lineare* (Rud.).

Shortly after, helminthologists discovered, upon a variety of birds, a curious parasite likewise inhabiting the trachea, but this time belonging to the order of nematodes, and especially characterized by the singular habit of permanent union of the sexes. Siebold[§] made it the type of a new genus—the genus *Syngamus;* later, however, yielding to the observations of Nathusius, he renounced his first idea and united this helminth with the strongyli in naming it *Strongylus trachealis*.[‖]

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[*] *Medical and Physical Journal* (1799), II, p. 204.

[†] Account of a species of fasciola which infests the trachea of poultry, with a mode of cure, *Trans. of the Wernerian Nat. Hist. Society*, I (1811), p. 195.

[‡] *Synops.* pp. 414, 415.

[§] *Archiv f. Naturgeschichte*, Wiegmann (1835), p. 1.

[‖] L. c., 1836.

After the creation of the genus *Sclerostoma* by Dujardin, in which this author unites the old strongyli possessing a mouth which is armed with a tough coriaceous capsule, Diesing placed in it the *Strongylus trachealis* of Nathusius under the name of *Sclerostoma syngamus*. Finally Dujardin[*] restored for this parasite the old genus *Syngamus* of Siebold, and gave it the old specific name of *Syngamus trachealis* of the same author.

Dujardin ascribes to the genus *Syngamus* the following characters:

> Worms ordinarily coupled in a permanent manner or by union of the integuments; the male, cylindrical, much smaller than the irregularly cylindrical female, with constricted neck and tail tapering to a point; head globular, large, supported by an internal corneous capsule; mouth large, irregularly rounded, with six or seven broadened lobes; pharynx provided with fleshy papillæ; integument folded or wrinkled without regular striæ. The male has a truncated tail, the latter provided with a membranous expansion which fastens itself to the integument of the female. The female has the tail conical, elongated; vulva situated anteriorly at the base of the constriction forming the neck; eggs large, elliptical.

The following, according to the same author, are the characters of the only species, *Syngamus trachealis*, which this genus includes:

> Body soft, colored bright red by a liquid interposed between the viscera. Male 4 to 4.5^{mm} (.157—.177 inch) long; .4^{mm} (.016 inch) wide; enlarged, obliquely truncated head about .7^{mm} (.028 inch) broad. Tail terminated obliquely by a convex, unilateral, membranous sac or bursa .25 to .3^{mm} (.009 to .012 inch) long, attached to the superior border of the vulva of the female and supported by 12 to 15 equal rays. Female 13^{mm} (.512 inch) long; .3 to 1^{mm} (.01 to .04 inch) broad, irregularly folded and wrinkled; head 1.3^{mm} (.05 inch) broad; tail resembling an elongated cone; anus 1.2^{mm} (.047 inch) from extremity; projecting vulva at the base of a neck 1.5 to 2^{mm} (.058 to .08 inch) long, inclined to one side; eggs smooth, elliptical, .087 to .093^{mm} (.0034 to .0036 inch) long, with a short terminal neck.

Dujardin found the *Syngamus trachealis* to the number of five pairs in the trachea of two magpies (*Corvus pica*) at Rennes. He was able to determine that even after maceration the male could not be separated from the female without rupture of the integuments.

This parasite has been found by Nathusius either in Germany or in England within the trachea of the following species: The swift (*Cypselus apus*), the starling (*Sturnus vulgaris*), the green woodpecker (*Picus viridis*), the pheasant cock (*Phasianus gallus*), and the black stork (*Ciconia nigra*), granting that it was the same species.

What relation exists between the two parasites of the bird's trachea spoken of above—the fasciola of Montagu, the cause of the gapes, and the *Syngamus* of Siebold?

Dujardin and Diesing regarded as entirely erroneous the classification among the distomes of the parasite found by Montagu in the trachea of birds affected with the gapes. This parasite was to them none other than the *Syngamus*, but as they did not enter into any details concerning the accidents which it is liable to produce, some doubts appear to have remained in the minds of French helminthologists concerning this assimilation. For we read in M. Davaine's treatise on Entozoa (2d ed. p. 37) the following statement concerning the parasites which cause the gapes among the Gallinae:

> These entozoa, which for a long time have been referred to the distomes, are probably identical with the *Sclerostoma syngamus*, a nematode worm, to which the permanent union of male and female has given a particular physiognomy which has deceived the earlier observers.

The word "probably," in the above extract well indicates that for M. Davaine there was as yet no certainty that the gapes was caused by the *Syngamus trachealis*; there was only a probability. Moreover, in the latest, fullest, and most noteworthy article which has appeared in France on the subject of helminthology as applied to domestic animals,* the author, M. Baillet, without saying a single word about the terrible disease, the gapes, with which in fact he does not seem to be acquainted, limits himself to noting the existence of *Syngamus* by the following sentence:

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[*] *Histoire nat. des helminthes in suites à Buffon.* Roret, Paris, 1845, p. 260.

> Before concluding the tribe of sclerostomes, we will mention the genus *Syngamus* (Siebold), a parasite of various birds which has been occasionally observed in the trachea of the cock and the hen.

This is all he says of this parasite. Up to the present, then, there have been only vague conceptions or none at all, concerning the pathogenic action of syngamus.† Even its natural history is poorly known, since in a remarkable monograph on a new nematode of the genus *Hedruris*‡ Prof. E. Perrier, citing incidently the helminths which present the peculiarity of a male united permanently to a female, says, concerning the parasite under discussion, page 6:

> Among the nematodes of the genus *Syngamus* the male lives attached to the female by means of a caudal sucking disk and *twines himself about her as does the male of Hedruis.*

This last statement italicized contains an error which proves that M. Perrier had not yet seen the syngames in the position which they occupy in the trachea, for the male is never coiled about the female, as we will show further on, and as we have enabled M. Perrier to demonstrate for himself.

We are now permitted to say, after having studied the gapes in the various pheasantries of central France, and the environments of Paris, where this terrible epizootic has claimed thousands of victims, that we know positively that the parasite which causes it, the so-called forked-worm, or red worm of the pheasant breeders, is none other than the *Syngamus trachealis*, and by no means a distome; we know that it corresponds entirely with the general characters traced by Dujardin and Cobbold, if we except a considerable number of anatomical and physiological details which we have to add or to rectify, and its migrations and habits which have thus far remained wholly undescribed. There was complete ignorance of its mode of development, reproduction, and its transmigrations. All these we have been able to follow experimentally or in the poultry-yards, and hence to deduce the most rational indications to combat the gapes successfully and to arrest its spread. Experience has fully confirmed our deductions.

ZOOLOGICAL AND ANATOMICAL DESCRIPTION.

We must, at first, rectify the diagnosis of the genus and species as given by the authors, because it appears to us faulty, especially in that which refers to the mouth-parts. We present the following diagnosis of the genus:

Mouth large, supported by a hollow, hemispherical, chitinous capsule, its background furnished with six or seven chitinous, cutting papillæ; border thick and turned back (*retroussé*), cut into six symmetrical lobes, united to the integument by its entire external face, and furnished by it with four equal membranous lips, which form a prolongation to the lobed border of the capsule. To this they are united by four bands, which attach the commissures of the lips to the four deeper notches between the lobes of the capsule. Female fixed by its mouth to the tracheal mucous membrane of its host; the male likewise attached by its mouth to the same mucous membrane and united immovably by its caudal bursa to the vulva of the female, around which it is soldered, as it were. The two spicules equal, contiguous, extremely fine, and very short. Ova provided with a valve at each end of the longer axis. The eel-like embryos are developed within the uterus of the female whence they emerge only at the death of the latter. Cuticle, with very delicate striæ, disappearing with age, but persisting in the cervical region.

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* Article *Helminthe, Dict. Vétérin.* of Bouley and Reynal, vol. III. Paris, 1866.

† According to Cobbold the *Syngamus* is the sole cause of the gapes.

‡ *Nouvelles Archives du Museum*, vol. VII., Paris, 1871.

Are there several species of *Syngamus?* Up to the present time helminthologists have agreed to admit but a single species, the *Syngamus trachealis* of Siebold; but the characters which they attribute to it differ in certain points from those of the species which we have studied as infesting the pheasants in France. Thus the latter attains twice the dimensions given by Dujardin. The head of the male, says this observer, is obliquely truncated, while in the species examined by us it is squarely terminal. The tail of the female, Dujardin continues, is an elongated cone and the anus 1.2^{mm} (.047 inch) from the extremity, while in the parasite of our pheasants the tail is either abruptly conical or rounded like a stump and pointed; in other words, it has the form of a cylindro-conical appendage, springing from the middle of the rounded posterior extremity (Plate I, Fig. 3); the anus opens at the base of this small tail, which is not more than .1 to $.2^{mm}$ (.004 to .008 inches) long.

Unless there was some error of observation, or some typographical mistake in the figures, or unless Dujardin had not seen the highest degree of development which the syngames attain, the parasite of the pheasant would constitute a distinct species, or at least a variety.

Without wishing to decide this question, which is only possible by making a direct comparison of the individuals found on different species of birds, we shall give the diagnosis of *Syngamus trachealis* after the species or variety which infests the pheasants before offering a detailed description.

Body cylindrical, becoming with age, in the female only, more or less sinuous or torulose; colored bright red by the coloring matter of the absorbed blood which tinges the nutritive fluid interposed between the organs.

Male 2^{mm} (.079 inch) long and $.2^{mm}$ (.0078 inch) broad at the beginning of union with the female, and reaching a length of 6^{mm} (.236 inch) and a breadth of $.5^{mm}$ (.02 inch) at the end of ovulation. Body always cylindrical, surpassed in its diameter by that of the head by $.2^{mm}$ to $.3^{mm}$ (.0078—.012 inch); posterior extremity slightly club shaped, inclined, oblique, terminated by a membranous bell-shaped sac or bursa, higher anteriorly than posteriorly, where it is cleft and notched along its entire height, supported by twelve simple rays, united to the vulva.

Female about 5^{mm} (.197 inch) long and $.35^{mm}$ (.0137 inch) broad at the beginning of copulation, attaining a length of 20^{mm} to 22^{mm} (·787—.866 inch), and a breadth at the middle of the body of 1.1^{mm} (.043 inch) at the end of ovulation; body at first cylindrical with delicately striated integument, becoming later more or less sinuous, torulose, and smooth, the striæ persisting only in the cervical region. Head 1^{mm} (.039 inch), broad at the period of complete development, surpassing the diameter of the neck by $.2^{mm}$ (.0078 inch), which is itself smaller by $.3^{mm}$ (.0118 inch) than the diameter of the middle of the body. Vulva springing from the base of an inclined neck, which is 1.5^{mm} to 3^{mm} (.059—.118 inch) long. Ova innumerable, smooth, elliptical, .085^{mm} to .09^{mm} (.0033—.0035 inch) long, and .05^{mm} (.002 inch) broad, each pole closed by a hood-like, hemispherical valve, which becomes entirely detached at the time of hatching. Embryos eel-like, developed in the body of the female, which sets them at liberty only by its death and the destruction of its body; at birth they measure .28^{mm} (.011 inch).

Habitat of the adults.—Trachea of pheasants.

We shall now study in detail the various parts of the body in the following order:

1. The general envelope of the body, consisting of the cuticle and the muscular layer lining it; 2, the digestive tube with its accessory parts; 3, the nervous system; 4, the system of excretory vessels; 5, the male and female genital apparatus as it exists in the most highly developed adults.

Body envelope.—The cuticle (Plate I, Fig. 5, *a*, *a*) is very thin, about .05^{mm} (.002 inch) thick, diaphanous, in appearance homogeneous, for we have been unable to distinguish several layers, as has been done with the larger nematodes. In young subjects it bears fine transverse striæ, but in old and united pairs of which the female is bearing eggs, and especially when these eggs contain well-developed embryos, the striæ of the trunk are completely effaced, but persist on the neck, where they can be best seen in the female, in which they are .087^{mm} (.0034 inch) apart, each fourth or fifth being deeper than the rest. Around the mouth the cuticle expands like a collar or gamopetalous corolla, with four equal rounded divisions forming four lips. At the same time it furnishes a broad margin to the thick and scalloped border of the buccal armature. In the male the cuticle goes to form the caudal, bell-shaped bursa, which is cleft posteriorly and longer anteriorly, the latter aspect being probably the true dorsal aspect of the worm. This bursa is supported by six simple rays on each side. It caps the hemispherically projecting vulva of the female and is united to it so intimately that even after the death of the worms and their maceration in water it becomes torn before it can be separated from the vulva.

The muscular layer which lines the internal surface of the cuticle forms four longitudinal bands, as among the other nematoid worms, two dorsal and two ventral, separated from each other by four linear intervals. These muscular tracts are very delicate and permit the internal organs to be seen through them. Only the superficial layer is distinguishable. It consists of longitudinal fusiform fibers (Plate I, Fig. 5, *b*) and is lined with parenchymatous cells, which may be regarded as a deeper muscular layer.

Digestive apparatus.—In the digestive tract three regions may be distinguished—the mouth, the œsophagus, and the intestine.

The mouth (Plate I, Fig. 4, *A*, *B*) opens on the anterior extremity of the body. It is surrounded by four equal symmetrical lips already described. At the four commissures of these lips may be seen four strong bands or nervures, which unite the membraneous labial circle to the coriaceous armature of the mouth. This armature, made up of brown chitine, has the form of a complete hemispherical capsule or cupule, the thick border of which is divided into symmetrical but unequal lobes. These consist of two large lateral lobes, each corresponding to a lip, two small anterior ones corresponding to the anterior lip and two small posterior corresponding to the posterior lip. At the opposite and symmetrical notches at the extremities of the large lateral lobes are inserted the bands or ligaments which separate or which unite the membranous lips. The bottom or background of the buccal cavity is a true pharynx, to which is attached the superior end of the œsophagus. It is pierced by a round aperture opening into the œsophageal cavity. On the periphery of this opening are disposed six, sometimes seven, radiating papillæ, hard like the capsule itself, with dorsal cutting edges. They are real fixed lancets, performing a function similar to those of the surgical instrument known under the name of bdellometer of Scarlandière.

The œsophagus (Plate I, Fig. 5, *c*) is relatively short, extending from the pharynx to the middle of the neck; it is club-shaped and very thick. Its lumen appears to us tetraquetral rather than triquetral, as among other nematoid worms. In fact, the pharyngeal insertion of the tube is crucial, *i. e.*, with four, not three, branches (Plate I, Fig. 4, A). The mucous membrane is surrounded by a longitudinal muscular layer, which, in turn, is enveloped by a layer of very stout radiating fibers, longer inferiorly. The whole is inclosed in a structureless membrane.

The upper extremity of the intestine into which the œsophagus opens is very wide. It is continued by a straight, wide, cylindrical tube, lined, in its entire extent, with brownish, distinctly nucleated cells, and terminates in a short oblique rectum, having the form of an inverted cone. The anus is situated at the base of the very short tail which measures only 1. to .2^{mm} (.004—.008 inch) in length. It appears to open most commonly on the dorsal aspect, that aspect which is opposite to the inclination of the head and neck or to the vulva. This is due to the spiral twisting of the female body when the uterus is laden with eggs. The anus of the male opens near the notching of the caudal bursa posteriorly. This shows that in the male also the ventral aspect is uppermost, which in the female is indicated by the vulva. In both sexes the anus is very small; and in fact an animal food, made up of the blood of the host, ought to furnish a very small quantity of solid waste.

Nervous system.—The nervous system of *Syngamus trachealis*, like that of the larger number of the higher nematodes, consists of a flattened ganglion forming a collar about the œsophagus, and giving off four quite symmetrical cords anteriorly and four posteriorly. The former pass to the mouth parts, the latter to the digestive and reproductive organs.

Secretory apparatus.—The most eminent helminthologists, among them Bastian, Schneider, and E. Perrier, have seen in certain nematodes secretory structures composed of utricles sometimes double, provided with a canal which opens on the skin in the middle of a papilla. These structures have been observed near the posterior extremity of the body in the male, and in the region of the neck in both sexes. We have sought them without success in the *Syngamus* of the pheasants. Once, however, we saw, quite distinctly, an oblique canal opening on the skin a little below the œsophageal nervous ring and arising from a glandular mass situated in the region, where, in Plate I, Fig. 5, we have shown the position of the longitudinal fusiform muscular fibers. Along the œsophagus and under the same muscular layers there is situated an elongated club-shaped gland, which opens at the base of the pharyngeal capsule (Plate I, Fig. 5, *d*). This is a true salivary gland; its walls are lined with ovoid, doubly-nucleated cells.

Reproductive apparatus: Genital organs of the male (Plate II, Fig. 7).—In the nematodes generally the testes consist of a long tube uniformly cylindrical in its whole extent from .1 to .2^{mm} (.004—.008 inch) in diameter. In the male syngame of the pheasants it presented quite characteristic differences from the known type. It is possible to see, through the translucent tissues of the body, and still better when the testicle has been forced out of the body of the worm, a large, abrupt expansion of the tube 1^{mm} (.04 inch) from its inferior termination. This bag-pipe-like enlargement gradually contracts anteriorly and continues as a cylindrical tube slightly narrower than at its commencement. At the middle of the worm's body it twines about the intestine, then redescends and terminates in a *cul-de-sac* near the posterior extremity. The disposition of this seminiferous tube may be better seen when, by a fortunate compression, or a patient dissection, it has been forced out of the body. The three portions of which it is composed may then be readily distinguished; the first as a vas deferens, the second as a vesicula seminalis, and the third (which coils about the intestine) as the testicle proper. The latter is filled with an opaque, amorphous substance, the contents of the vesicula seminalis and the vas deferens being likewise opaque but segmented into granular corpuscles of very varying forms, having each a nucleus of .01 to .03^{mm} (.0004—.0012 inch) in diameter. These are the spermatozoids. The vas deferens, about .075^{mm} (.003 inch) in diameter, opens at the posterior extremity of the body in the center of the caudal bursa, between two very small, short, and nearly straight spicules, the extremities of which rest immovably in the vagina of the female. The vesicula seminalis, enlarged in the form of a pear, has its walls made up of muscular fibers which are all obliquely placed and inserted into a longitudinal raphé like the barbs of a feather into the shaft. The object of this arrangement undoubtedly is to cause the expulsion of the spermatozoids and their projection into the vagina of the female, the long duration of this function requiring a special and powerful apparatus.

Female genital apparatus (Plate II, Fig. 6).—As in almost all nematodes, the female generative organs comprise a uterus with two long branches narrowing abruptly into a tubular portion, the ovary proper. We have not been able to discover a bag-pipe-like swelling near the commencement of the ovary which E. Perrier has seen in the *Hedruris armata*, and which he calls the copulation pouch (vesicula copulatrix). Neither this pouch nor anything similar to it exists in the syngame.

The vulva, as has been stated, is a small opening pierced through the summit of a hemispherical papilla which is permanently covered by the caudal bursa of the male. The vagina, the canal which penetrates the papilla, is very narrow. Lodging the spicules of the male it serves as a passage for the spermatozoids which the male pours into it during his entire adult existence. It will be readily understood that it never fulfills the function of oviduct, since the inseparable union of male and female renders the discharge of ova through the vagina impossible.

The vagina is continued into a short, enlarged uterus, about .6^{mm} (.024 inch) long and broad, which divides into two long cylindrical horns, having a diameter of .3^{mm} (.012 inch) at the base and .25^{mm} (.009 inch) at the apex. They are about three times as long as the intestine, about which they coil in the most capricious windings. The uterus and its horns are filled with ova, the development of which proceeds with the age of the worm, as we shall see further on. Each horn at its apex contracts abruptly into a short cone, and is continued by a small tube about .05^{mm} (.002 inch) in diameter, which might be likened to a Fallopian tube. After a distance of 3^{mm} (.118 inch) these tubes gradually dilate into tubes of twice their diameter, filled with spherical, granular corpuscles, compressed and crowded together in one or two rows. These are the ovules, the tubes containing them, the ovaries. As long as the uterine horns, these tubes are wound in a thousand different ways about the intestine, then contract each into a tube as narrow as the Fallopian tubes (or oviducts), containing only amorphous matter, and lastly terminate in a *cul-de-sac* devoid of dilatation or enlargement.

Amongst the ova filling the uterus and its horns, we have determined the presence of spermatozoids closely resembling those contained in the vesicula seminalis and the vas deferens of the male, but we have not succeeded in seeing them elsewhere. We believe that the fecundation is effected in the uterine horns near the ovarian extremity upon the ovules brought there by the Fallopian tubes, since there is here no organ similar to the vesicula copulatrix, which E. Perrier has pointed out in the *Hedruris armata.*

EMBRYOGENY AND DEVELOPMENT.

It has already been stated that the narrow terminal extremity of the ovary is filled with a finely granular, amorphous, opaque, and homogeneous substance. On approaching the coiled portion of the ovary this granular matter is seen to unite into spherules, which are the ovules proper. They are ranged in a single row owing to the narrow tube, the internal diameter of which they almost fill up. In the wider portion of the ovary they range themselves in two or three rows. Near the oviduct (Fallopian tube) they first become slightly ovoid, with a long diameter of .08^{mm} (.003 inch), and they possess a distinct germinal spot and vesicle. Still without shell or distinct envelope, they are led, one by one, through the oviduct into the corresponding uterine horn, where they find themselves in contact with the spermatozoids, and where they become inclosed in a shell. When this is completed, and the egg consequently perfect, it presents the form of an ellipsoid, with a long diameter of .09^{mm} (.0035 inch) and a short one of .05^{mm} (.002 inch). The egg is not truncated nor provided with a neck at each extremity, as is the case with many nematodes. There is, on the contrary, at each pole a thickening, hemispherical externally and almost flat within (Plate II, Fig. 8, A, B, C). This is an actual cover, detaching itself completely when the embryo emerges. Only the empty ovum, therefore, is really truncated at its two extremities.

In the uterine horns the ova undergo complete segmentation. Their vitellus divides into 2, 4, 8, 16, &c., small spheres, which assume the mulberry form (Plate II, Fig. 8, A). The development proceeds in the lateral regions of the egg (Plate II, Fig. 8, B), and at its close the embryo may be seen rolled up in the form of a circle or a figure of eight. The egg is now .1^{mm} (.004 inch) long and .06^{mm} (.0024 inch) broad.

But it is not to be supposed that all the developmental phases of the ovum can be followed out in every syngame. Only in case of the largest specimens can this be done by examining successively the genital organs of the female, from the extremity of the ovaries to the body of the uterus after they have been taken from the body and well spread out. It is also possible to trace the series of successive transformations which the ovule undergoes from the embryonic to the perfect state by examining a series of females from the moment of their sexual union with the male to that of their greatest development. Thus in the syngames recently conjugated, at a time when the female is scarcely 5^{mm} (.2 inch) long, only spheroidal ovules are found in the uterus and its appendages, which are very short, but slightly developed, and not distinct from the ovaries, their diameters being the same. When the female has reached a length of 1^{cm} (.4 inch), the uterus and its horns, now quite distinct, contain eggs fully formed and inclosed in a shell, but the vitellus is not yet segmented. When the body is 15^{mm} (.59

inch) long the vitellus is already segmented, and has even passed beyond the morula stage, as many of the eggs, particularly in the body of the uterus, reveal the embryo in process of development. Finally, when a length of 20 to 22^{mm} (.787–.866 inch) has been reached, eggs containing fully formed embryos, rolled up and moving within their narrow prison, are observed in the two divisions of the uterus. At this period they may be forced out of the shell by pressure between two glass slides; the covers at the extremities detach themselves completely and the embryo emerges through either opening. When it leaves the egg spontaneously, an act we have frequently observed in the water, the cephalic extremity always emerges first.

The embryo, on leaving the egg, exactly resembles an agamous anguillula (Plate II, Fig. 8, D). It is about .28^{mm} (.011 inch) long, and has a diameter of .013^{mm} (.0005 inch) at the middle of the body. The obtuse anterior extremity reveals a punctiform mouth, opening in the middle of a papilla and continued into an œsophagus which occupies the cephalic third of the body (Plate II, Fig. 9), and whose cavity is distinguished as a very fine median line. This portion of the body is clear; the remaining two-thirds is filled with granulations or fine globules. The tail is conical and elongated.

The embryos never leave the egg within the living body of the mother, however complete the development of both may be. Only by the death of the female and the destruction of its body are the ova placed at liberty. The embryo will then emerge if the medium offers favorable conditions. These are moisture and a temperature of at least 20° C. (68° F.). These facts we have frequently demonstrated by experiment and in other ways. We have even found still attached to the trachea of pheasants destroyed by the gapes couples of dead syngames, with the soft, flaccid body of the female, 24^{mm} (.945 inch) long, opened in several places by the commencing process of maceration, through which a large number of eggs had already escaped. It still contained many of them, each inclosing a fully developed, very active embryo, but there was not a single empty egg or free embryo in the entire cadaver.

We have subjected the eggs to various conditions in order to determine those most favorable to the hatching of the young. 1. When in a dry medium, as in sand, their contents dry up more rapidly in proportion to the elevation of temperature. 2. In a moist state they preserve their vitality for months, even for a year, without any perceptible modification of their contents, if the temperature is kept below 15° C. (59° F.). Under these conditions the contents finally undergo fatty degeneration and are dissolved. 3. If, while in a humid state, the temperature be raised to 20° C. (68° F.) or better, to 25° C. (77° F.), the embryo within the egg moves and turns about and finally escapes by pushing away one of the coverlets.

The combined conditions of moisture and warmth are powerful enough to bring about the development of the embryo and its escape from the egg, in which at first no trace of it can be distinguished, and which contains only the vitellus. In the water contained in crystallizing dishes, small enough to be placed on the stage of a microscope, we have studied day by day the formation of the embryo during the month of July of this year, when the temperature maintained an average of 25° C. (77° F.). We have determined that in presence of these conditions twenty-eight to thirty days suffice for the development of the embryo and its escape from the shell.

The embryos or larvæ live in the water, where they swim about in a serpentine manner like the anguillulæ (vinegar eels, &c.). At a temperature of 20° or 25° C. (68°–77° F.) we have been unable to keep them alive for more than eight or ten days, whilst at a lower temperature they lived for many months, almost a year. During this time they molt, the tail becoming less elongated, and assuming the form of a short cone (Plate II, Fig. 10). When the hatching has been delayed from insufficient warmth, and the embryo finally escapes from the egg, it leaves within the shell an envelope. This fact seems to prove that the molt, which takes place normally one or two days after birth, occurs in the egg itself when birth is retarded. In the experiment-glasses larvæ with short tails were often seen moving among those with long tails. The former were simply older than the latter.

The following questions now arise: Does the larva molt a second time before assuming the adult form, and what are the ways and means employed by it to reach the only place where adult and paired syngames are found—the trachea of birds?

Some species of *Sclerostomata* presents a nymphal phase, during which the young parasite is provided with an almost complete buccal armature, and lives, rolled up and encysted beneath the mucous membrane to which it attaches itself in its adult state. Repeated investigations have failed to reveal anything analogous in the syngame of the pheasants. We have every reason to believe that the nymphal stage, no doubt very short and active, is passed in the air-sacs and pulmonary bronchi, which, as is well known, intercommunicate very largely in birds, and which the larvæ may readily reach by traversing the intestinal or œsophageal tunics after escaping from the ingested eggs. We also believe that the parasites very soon after reach the trachea, to become adult, pair, and attach themselves. The following are the facts upon which this opinion is based:

1. The larvæ of Syngamus, according to our observations, do not develop well, nor will they leave the egg and become vigorous excepting in a moist and warm medium, approaching the conditions offered by the interior of a bird's body.
2. In a young pheasant, dead from the gapes, we found in the mucus obtained by scraping the lining membrane of the œsophagus, a large number of eggs of syngames with the shell opened and abandoned by the embryo. We have preparations to demonstrate this fact.
3. In the serous fluids which lubricate the walls of the air-sacs, more particularly those in relation with the duodenum, we have found in the case of young pheasants attacked with the gapes very active larvæ, almost twice as large as those just emerging from the egg, seeking their way.
4. In the cellular peritracheal tissue, in the neighborhood of the crop of one of the young pheasants referred to above, we found, stretched out parallel to the trachea, a young female syngame, already colored red, 5^{mm} (.2 inch) long, with the mouth formed like that of the adult, and even sexually matured. We think that it was a syngame which, having been delayed in the migration, failed to reach the mucosa of the trachea in due time and now could no longer do so, because the adult structure of the mouth-parts presented an impediment to its march across the tissues.
5. In the inclosures of M. de Janzé, at Gournay (Eure), which were desolated last year by the gapes, and which have presented this year some cases of this disease, the following fact has often been observed and verified by M. de Janzé himself: The young pheasants affected with this malady frequently expel, in a fit of coughing, plump, fat syngames full of eggs. The other fowls near by consume with avidity the

worms thus ejected, which they, no doubt, regard as earth-worms, or the red larvæ of the large tipulæ which resemble them, and of which they are very fond. Two or three weeks later these young pheasants are sure to present symptoms of the malady—the slight, aborted hissing cough, which is so characteristic, and the gaping, which has gained for this disease its English name.

6. For the purpose of verifying experimentally the accuracy of the facts related above, the authenticity of which, however, did not give rise to any doubt, we fed to a female parrot, on the 7th of August, four pairs of large syngames. We had just received from Mme. de la R—— de Montmirail some young pheasants, dead from the gapes, from which we obtained an ample number of syngames; the parrot being the only subject we had for experiment at the time. On August 28 this bird began to cough and to gape. On September 10 it died, suffocated by numerous syngames which we found, at the autopsy, crowded in the trachea.

Considering the large number of eggs—several thousand—which a cadaver of the female syngame contains, and the relatively small number of parasites—about thirty or more pairs—which reach their destination, or, in other words, come to maturity, we may form an estimate of the prodigious number of larvæ which die on their way or never succeed in finding it. It is, moreover, a law of nature, especially true of parasites, that the number of eggs laid is larger in proportion as the chances of destruction during the earlier period of existence are more numerous.

The great variation in the size, and hence in the age and the degree of development, noted among the syngames attached to the trachea of a bird shows that there are ordinarily several successive infections or ingestions of eggs at intervals more or less extensive. This fact may also be due to the circumstance that the conditions favorable to the development of the parasite have not been the same for all.

The feeding of healthy pheasants upon syngames filled with eggs, which have been ejected by pheasants suffering from the gapes, is not the only means by which this disease may be propagated. The observations which we have made concerning the vitality retained by the eggs of the parasite when in a moist medium—a medium in which the embryos are born and developed if the temperature reaches a suitable height (20°-25° C.)—prove that the ingestion of water and liquid or pasty aliments, containing these embryos or eggs, furnishes two other means of infection perhaps more active than the first. In every case the only media necessary for the propagation of epidemics of the gapes are food and drink contaminated with the eggs or embryos, and the birds themselves when affected with the disease, as they are then the source of an abundant emission of eggs of the parasite. No other animated medium, neither adult insect nor larva (the larvæ of ants, for example, which are a constant element of food for young pheasants, and which have been suspected with some appearance of truth), nor any mollusk, in short, can be incriminated.

MEANS OF DESTROYING THE SYNGAME AND OF ARRESTING EPIDEMICS OF THE GAPES.

The disasters caused by the parasite above described in the parks devoted to the rearing of pheasants, point out the extreme importance of finding rapid and effective means of arresting the spread of this destructive worm.

A remedy, common in England, consists in mixing the grains which are to be fed to the diseased birds with urine instead of water. Montagu, who tried this remedy without having any faith in its efficacy, was surprised at the success which he achieved, and which proved to him that it was not without utility. It is probable that the ammoniacal emanations arising from the urine are poisonous to the red worm or its embryos.

Wiesenthal relates that in America a hen's feather is stripped of its barbs to near the point, introduced into the trachea and rotated like a brush to detach the worms. We strongly question the efficiency of this practice; in the first place, because we know from experience that the worms are too firmly attached to be removed by the friction of the barbs of a feather. Should they be detached, however, they would only be pushed to the root of the trachea, where, forming a ball, they would augment the obstruction in the tube and thus bring about more promptly the death of the bird. On the other hand, the diameter of the trachea of a young pheasant from five to six weeks old, being scarcely equal to that of the shaft of a hen's feather, will not permit the introduction of the latter. Cobbold,* on the contrary, believes in the efficiency of this method, and adds that this efficiency may be increased by impregnating the feather with a germicide substance. Bartlett, who used salt for this end, or a weak infusion of tobacco, informed him that the essence of turpentine also had given excellent results. Cobbold adds with reason, that unless great care be exercised with this method the birds may be seriously injured.†

These means, at once mechanical and medicinal, have been suggested several times and varied in different ways. One of our correspondents informed us that he had cured pheasants of the red worm by removing the parasites with a small rod and pouring into the mouth of the birds a few drops of Fowler's solution. Another pretends to have removed the parasites with a piece of copper wire, which had one end curved like a handle and dipped into *oleum hypericum* (red oil). We do not doubt that they could have succeeded in thus removing red worms lodged in the pharynx, but we do not believe that they could have extracted worms by this method from the root of the trachea near the bifurcation of the bronchi, where they are most frequently lodged; for it is actually impossible to employ a rod, and above all, a metallic wire curved into a hook, as it would undoubtedly tear the trachea. The fact that young pheasants, and more frequently adults, sometimes recover spontaneously from the gapes, may have given rise to their apparent success. This happens when they are affected by only a small number of parasites, which may go through the phases of their development to their death without producing suffocation. This is the only mode of fatal termination, and it requires a certain number of parasites, from twenty to thirty couples for adult, and from five to ten for young pheasants. In these cases the disease is cured in spite of, and not because of, a certain mode of treatment.

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* *Parasites:* London, 1879; p. 445.

† Cobbold's exact words concerning this method are as follows (*loc cit*):

"First. The simplest plan consists, as Dr. Wiesenthal long ago pointed out, in stripping a feather from the tube to near the narrow end of the shaft, leaving only a few uninjured webs at the tip. The bird being secured, the webbed extremity of the feather is introduced into the windpipe. It is then twisted round a few times and withdrawn, when the worms are found attached. In some instances this plan succeeds entirely.

"Secondly. The above method is rendered more effectual when the feather is previously steeped in some medicated solution which will destroy the worms. Mr. Bartlett employs salt for this purpose, or a weak infusion of tobacco; and he informs me that the simple application of turpentine externally is sufficient to kill the worms. It should be borne in mind that the bird itself may be injuriously affected by these drugs if they are carelessly employed." Note that the turpentine is to be used externally.—TRANSR.

One of the most rational methods of treatment has been pointed out by Montagu, who did not stop with the common method above mentioned, but who obtained much success with the following means combined: Removal from the infected places, complete replacement of the former aliments by new ones, in which hemp-seed and fresh grass figure prominently; finally, for drink, an infusion of rue (ruta) and garlic, instead of ordinary water.

The efficacy of the garlic was demonstrated to us under the following circumstances: The pheasantry in the forest of Fontainebleau was laid waste by the gapes in 1877 and 1878. This malady, which we studied on the site of its activity, was arrested and completely driven out by feeding the pheasants with a mixture consisting of hard-boiled eggs, boiled beef's heart, the crumbs of stale bread, and salad. These ingredients were chopped, pounded, and thoroughly mixed so as to make a paste. To this paste was added pounded garlic in the proportion of one clove or bulb to ten pheasants each day, the garlic being thoroughly distributed through the paste. This mixture was relished very much. Great care was bestowed upon the drinking vessels; the very pure water used was renewed twice a day. The same treatment was successful in the several inclosures belonging to the country-seats in the neighborhood of Fontainebleau and Melun. A large number of correspondents to whom we suggested it were fully satisfied in having applied it. We also learned that the pheasants occasionally refused the garlic, and one of our correspondents informed us that he had succeeded in making them take it by preparing a real garlic salad; for he had accidentally observed how the animals which had refused the garlic paste cast themselves voraciously upon a garlic salad which was not intended for them.

We can readily explain the virtue of garlic, known from time immemorial as an excellent anthelmintic, as it is volatile and is eliminated by the respiratory passages, reaching, in this way, the trachea, where the syngames are lodged. The proof that the essential and volatile principles of garlic are eliminated by the lungs is daily furnished by those persons who, like the inhabitants of the south of France, are fond of this condiment. The odor of their breath betrays them immediately.

Besides garlic, we have experimented with another substance, which, like the former, has the advantage of being a strong-smelling vermifuge and more stupefying than ether (which might also be employed), properties which enhance its parasiticide powers. We refer to assafœtida, which we have used as a powder with an equal part of yellow pulverized gentian, mixed with the paste which is fed to the pheasants, in the proportion of 50 centigrams (about 7½ grains) per head each day. As a complement to this treatment we have added to each liter (or quart) of drinking water the following solution: Salicylic acid, 1 gram (about 15½ grains); distilled water, 100 grams (about 3½ fluid ounces).

The use of the salicylic acid, the toxic power of which upon the embryos of syngames we have recognized experimentally, had for its sole object the destruction of those embryos which might be present in the drinking water of the young pheasants.

This treatment we have employed in the parks of Baron Rothschild, at Rambouillet, which were ravaged by the gapes in a manner so disastrous that up to 1,200 young pheasants were found dead each morning. A letter from the baron's steward, dated September 7, 1879, testifies that the treatment has fully succeeded in arresting, in a few days even, the epidemic.

We will conclude these suggestions by stating that it is alway beneficial and even indispensable to disinfect the soil of the inclosures after having transferred the young pheasants to a virgin soil. One of the best means of destroying the eggs and embryos which may possibly exist on the soil of the contaminated inclosures, consists in sprinking it with water containing in solution a sufficiently large quantity of salicylic or sulphuric acid, one gram (15½ grains) to a liter (about 1 quart) for example.

Great care should also be taken to isolate the sick birds on the first appearance of the symptoms of the disease, and to keep them closely confined till complete and well-confirmed recovery. The cadavers of dead birds must be buried deep, or it were even better to burn them.

SUPPLEMENT.

In the investigations which we have made concerning the development of *Syngamus trachealis*, and which are reported in the preceding memoir, written about twenty months ago, we pointed out that the eggs ejected during the coughing fits hatch in the water, and that the embryo, resembling an anguillula, may live in this medium for many months, because we have kept some alive almost a year in a low temperature. The birds are infected by drinking the water containing these embryos. But how are they developed in the body of birds, and in what way do they reach the trachea, where they are found, in the adult state, fixed to the mucous membrane like leeches, the two sexes united in a permanent manner and the females crowded with eggs?

In the preceding memoir we stated that we had every reason to believe that the nymphal phase, unknown to us, was passed in the air-sacs and bronchi, and that later on the worm reached the trachea where it became adult. We offered as a proof of this hypothesis the discovery of embryos of syngames, in every respect similar to those which we had obtained from the hatching of ova, in the air-sacs of several young pheasants killed by the gapes.

There was, therefore, only a presumption, well founded, it is true, of the existence of the nymphs in the bronchi of the pheasants. At present it is no longer a presumption but a certainty. At the autopsies, lately made, of two red partridges, killed by syngames, we met the nymphal form in the pulmonary tissue itself, rolled up in the bronchial dilatations. (Plate II, Fig. 11.) It is cylindrical, very elongated, about 1.6 to 2^{mm}. (.063 to .079 inch) long, and .04 ^{mm}. (.0016 inch) in diameter. It is, consequently, ten times as large as the embryo when it leaves the egg, and one-tenth as large as the adult worm at the period of its greatest development. The armature of the mouth is already cupulate or cup-shaped, but still without color, border, and lobes. The muscular œsophagus is very long and cylindrical. The intestine, which extends in a straight line from the termination of the œsophagus to the anus, fills almost the entire body, and is already colored red; near the anterior third of the body may be seen a fleshy thickening, which sends a prolongation forward beyond the posterior extremity of the œsophagus, and one, longer than the first, backward toward the caudal extremity. This is the rudiment of the sexual organ.

This discovery of the nymph enables us to say that all the developmental phases of *Syngamus trachealis* are now known. The only two media which this parasite inhabits during its entire existence are the water or moist earth during its embryonal condition, and the respiratory organs of its victim during its nymphal and its adult phase. It is, therefore, developed without the aid of any other medium than the water, corresponding in this respect to the immense majority of verminous parasites.

CONTAGIOUS PLEURO-PNEUMONIA IN NEW JERSEY.

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The position of New Jersey in its relation to the seaboard, to the two great cities of New York and Philadelphia, and to the transportation and delivery of live stock, identifies it most intimately and unavoidably with all that relates to the communicable diseases of animals. Also as a grazing State and by reason of its own large city population and its relations to cities near by in other States, it has inducements to furnish a milk and butter supply much beyond what its gross acreage would indicate. The number of cattle, as stated in the last census, was 221,864, of which 152,078 were milch-cows. This number is constantly on the increase.

The first record we have of contagious pleuro-pneumonia in New Jersey is thus stated by Dr. C. Michener, now of Pennsylvania. It first broke out (see 2d Report State Board of Health, 1878), in 1847 in the herd of Mr. Thomas Richardson, he first finding it among his imported stock, and, knowing its malignancy, immediately resorted to occision at a great sacrifice, thereby stamping it out. Again, in the summer of 1855, six years before it made its appearance in Massachusetts, J. L. Jacobus bought twenty head of cattle in New York, which he turned into pasture, three miles from Chatham, N. J. In about three weeks, in going to look after the herd, he found two had died and three more were very sick. The remaining fifteen did not develop the disease. At about the same time Dr. Munn, of Chatham, N. J., purchased some cattle from an apparently healthy lot, which he yarded with his other stock, when the disease made its appearance among his other cows, some of which soon died. From Dr. Munn's herd the disease was communicated to that of Mr. Lunn, who also lost several animals. Mr. Abraham Johnson, living near Newark, also purchased some cattle in New York which developed the disease in a short time after he brought them to his farm.

The following further statement is from a letter of General N. N. Halsted, of Newark, Essex County, as made to the Agricultural Department (1878), "Information in relation to diseases prevailing among swine and other domestic animals":

> In 1859-'60, the first years of the appearance of pleuro-pneumonia in this State, I had the honor of being president of the State Society, and with Governor Olden's assistance and the generosity of some few of the members and officers of the association, we made an exhaustive examination into said disease, bought the diseased cattle, quarantined them, killed some, and made, through our surgeons and veterinary surgeons from New York and this State, a careful autopsy of several we killed and many of those that died. The result of these investigations was published in the annual report of the State society. We went to Boston and made a thorough and careful examination there, and decided that the disease was an imported one.
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> The disease was brought into our State by Mr. Johnson, who bought six calves from the swill-milk stables in Brooklyn, N. Y. These brought the disease to his herd. The society stopped it there, and we had no more of it until our Union County

farmers brought some more swill-milk stable animals, and being sellers of milk, kept the matter quiet, or hid it from the officers of the society, until the whole neighborhood was infected. This has been stamped out by a rigid quarantine and the use of carbolic acid, used as a disinfectant, and by the animal's inhaling it. They have some of it now in Burlington County, produced from the same cause, which is being eradicated by the same means.

It was on the farm of this Mr. Abraham Johnson, near Newark, N. J., that, according to Dr. Michener, Dr. C. C. Gryce, of New York, in the presence of several physicians, made the first expert *post-mortem* examination of this disease. At that time and for a considerable time subsequently there was no systematic investigation of the disease, and as most of the veterinarians had never seen it we had only fragmentary evidence as to localities where it existed. Robert Jennings, V. S., records its existence in Camden and Gloucester Counties, New Jersey, in 1859, and its introduction into Philadelphia in 1860. It spread to "The Neck" in the southern part of Gloucester County (Gamgee's Report, p. 20), killing from 30 to 50 per cent. of infected herds, and appeared in 1861 in Delaware and Burlington Counties, New Jersey. Those who from time to time have had oversight of it in New Jersey are also able to refer to other cases in other localities where there were in single herds unusual losses of cattle, and the description of the *post-mortem* appearances of which, by butchers or intelligent farmers, leads to the strong impression that the disease was pleuro-pneumonia.

Up to the period, say, of 1860–'61, Long Island, and especially Brooklyn, must be considered as the center of the disease. In addition to the German cow of 1843 or 1850, a fresh supply of the lung-plague poison reached Brooklyn from England in the system of an imported cow. The Massachusetts outbreak (July, 1859), was independent of the Long Island center, as it was from a direct importation from Holland to Boston.

There is no evidence that any center of contagion was established in New Jersey by the cases of Mr. Thomas Richardson in 1847.

The cases of Mr. Johnson, near Newark, in 1859, were directly traceable to Brooklyn purchases. So far as stated in a general way by Dr. Robert Jennings, it does not appear that the origin of the cases said to have occurred in Camden County in 1858, and in Gloucester County in 1859, was distinctly traced. The cases are stated as if antecedent to those in Philadelphia in 1860. Mr. J. E. Hancock, of Burlington County introduced the disease in his herd by purchases in Philadelphia in 1861.

After this we have found no authentic reference to the disease until it is announced as being in Ocean and Camden Counties in 1871, where it caused great loss; in Essex and Union Counties in 1872; in 1873 causing in Burlington County a loss of about 100 head, and in 1877 in a Jersey herd near Burlington. In 1878 it was in herds in Hunterdon County, near Clinton.

Two facts are very significant in all the cases of outbreaks of which we have been able to secure any particulars. The disease did not seem to spread far from any single herd after it was known to exist in it, even when introduced, and when, a year or more after, a second outbreak occurred, it generally came by the purchase of an animal and its introduction into some new herd. While the fact of its being a contagious disease was undoubtedly established, it was also evident that it belonged to that kind of contagion which is not carried far through the air, but is either inhaled or communicable only at short distances.

While the disease attracted the attention of the State Agricultural Society, no very decided means were taken for its eradication, except, perhaps, in Essex County, and in the vicinity of Newark, where inoculation was early introduced by a German veterinarian, and where it was, no doubt, found effective in limiting the malady in special herds.

Some representations as to its prevalence were made to the legislature of New Jersey during 1878, so that March 13, 1879, an act was passed, entitled "An act to prevent the spread of contagious or infectious pleuro-pneumonia among cattle in the State of New Jersey." The act is as follows:

1. *Be it enacted by the Senate and General Assembly of the State of New Jersey*, That whenever it shall be brought to the notice of the governor of this State that the disease known as contagious or infectious pleuro-pneumonia exists among the cattle in any of the counties of this State, it shall be his duty to take measures to promptly suppress the disease and prevent it from spreading.

2. *And be it enacted*, That for such purpose the governor shall have power, and he is hereby authorized, to issue his proclamation stating that the said infectious or contagious disease exists in any county or counties of the State, and warning all persons to seclude all animals in their possession that are affected with such disease, or have been exposed to the infection or contagion thereof, and ordering all persons to take such precautions against the spreading of such disease as the nature thereof may, in his judgment, render necessary or expedient; to order that any premises, farm or farms, where such disease exists or has existed be put in quarantine, so that no domestic animal be removed from or brought to the premises or places so quarantined, and to prescribe such regulations as he may judge necessary or expedient to prevent infection or contagion being communicated in any way from the places so quarantined; to call upon all sheriffs and deputy sheriffs to carry out and enforce the provisions of such proclamations, orders and regulations; and it shall be the duty of all sheriffs and deputy sheriffs to obey and observe all orders and instructions which they may receive from the governor in the premises; to employ such and so many medical and veterinary practitioners, and such other persons as he may from time to time deem necessary to assist him in performing his duty, as set forth in the first section of this act, and to fix their compensation; to order all or any animals coming into the State to be detained at any place or places for the purpose of inspection and examination; to prescribe regulations for the destruction of animals affected with the said infectious or contagious disease, and for the proper disposition of their hides and carcases, and of all objects which might convey infection or contagion; provided that no animal shall be destroyed unless first examined by a medical or veterinary practitioner in the employ of the governor as aforesaid; to prescribe regulations for the disinfection of all premises, buildings, and railway cars, and of all objects from or by which infection or contagion may take place or be conveyed; to alter and modify from time to time, as he may deem expedient, the terms of all such proclamations, orders, and regulations, and to cancel or withdraw the same at any time.

3. *And be it enacted*, That any person who shall transgress the terms or requirements of any proclamation, order, or regulation issued or prescribed by the governor under the authority of this act, shall be deemed guilty of a misdemeanor.

4. *And be it enacted*, That all the necessary expense incurred, under direction or by authority of the governor in carrying out the provisions of this act shall be paid by the treasurer upon the warrant of the comptroller on being certified as correct by the governor.

5. *And be it enacted*, That this act shall take effect immediately.

Approved March 13, 1879.

This law placed large discretion in the hands of the governor of the State, who at once proceeded to give it the most ample facilities for execution. Mr. Wm. H. Sterling was promptly placed in charge and commenced operations March 15, 1879. A system of inquiry throughout the State was at once instituted, and several veterinary practitioners placed on duty. Arrangements were made for searching out all cases of the disease in the State, as well as for examining all cattle brought into or passing through the State. The fact that New York already had such a provision, and that the ferry companies on the Delaware responded to the plan, enabled the inspectors to watch all transportation. An order under date of March 26, 1879, that all inoculation of cattle should cease, shows that this method was being relied upon in some parts of the State. The disease at this time was regarded as existing chiefly in the counties of Hudson and Bergen, as appears from a letter of Mr. Sterling dated March 27, 1879. March 28, it appears that a veterinarian had inspected the abattoirs at Communipaw and Jersey City and found all the cattle seemingly healthy, but in Newark and vicinity writes that he found a number of cases which he quarantined. Dr. Holcombe reported cases at Leonia and Ridgefield in Bergen County; Dr. Rogers, at Wellington and Sterling, Warren County, and also a herd was reported as inspected at Perth Amboy, Middlesex County. April 1, 1879, Bergen and Hudson Counties were placed in quarantine. Up to April 7, 1879, there had been inspections, also, in Bergen and Hudson Counties, and 25 cattle had been reported as having pleuro pneumonia and 8 parasitic disease. Of the 25 cases, 8 were in Hudson County. Further investigation cast some doubt on the cases in Bergen County. From April 7 to April 11, 137 herds were examined in Bergen County, and no sick animals found. In Hudson County 56 herds were examined and 23 sick animals found. Two sick animals in different herds were also reported in Union and Essex Counties.

The report of April 16 shows 23 cases in all in Bergen County in 8 herds. At the same time 28 were reported sick in Hudson County, the number of herds not being stated. The report of April 16 says that 33 herds in all, containing 259 head, of which 73 were diseased, were in quarantine. Of these, 21 herds were in Hudson County. Thus far 2 cows had been killed—one on the farm of Charles McMichael, at Leonia, Bergen County, and one belonging to Mr. Spargleburg, in Jersey City. April 26, 57 stables were reported in quarantine, in which were 114 diseased cattle. Fourteen had been killed at the cost of the State. A report of May 10, 1879, gives a total of 67 herds in quarantine and 178 sick. May 17, 1879, 76 herds, of 499 cattle, were in quarantine, with 185 sick. May 24, in the same year, 80 herds, of 521 cattle, were in quarantine, with 196 sick. Over 60 head of these were in Hudson County, with a few in Bergen, Middlesex, Mercer, Monmouth, Essex, and Union.

May 31, in the same year, there were 82 herds in quarantine, and 204 sick cattle.

June 7, 1879, 78 herds were in quarantine and 186 cattle sick, with 56 suspicious cases to be reinspected.

June 13, 1879, 81 herds in quarantine, containing 610 cattle, with 220 sick. A herd in Hunterdon County and one in Gloucester County were affected. June 21, 1879, 79 herds in quarantine, of 586 cattle, with 213 sick.

The southern part of the State was also found infected, the infection having come from Philadelphia. June 28, of the same year, 85 herds were in quarantine, with 653 cattle, and 242 sick. July 5, 1879, there were 92 herds in quarantine, of 681 cattle, with 245 sick. Inspections were being made in Middlesex, Mercer, Monmouth, Morris, Hudson, and Hunterdon Counties, and of special cases in Camden and Gloucester Counties.

July 19, 1879, there were 96 herds in quarantine, of 765 cattle, with 264 sick. Two herds in Burlington County were added to the list. On July 7 Essex County was placed in quarantine. July 26, 1879, there were in quarantine 99 herds, of 800 cattle, with 265 sick, and inspections were being made in 6 counties.

August 2, 1879, 101 herds were in quarantine, of 796 cattle, of which 271 were sick, and 7 counties were undergoing inspection.

August 9, 1879, 107 herds were in quarantine, of 833 cattle, of which 288 were sick.

August 8, 1879, 271 sick cattle had been condemned and 106 destroyed, mostly by their owners without compensation.

August 16, 1879, 111 herds were in quarantine, of 878 cattle, with 319 sick.

August 23, 1879, 112 herds, of 874 cattle, were in quarantine, with 325 sick.

September 5, 1879, 109 herds, of 870 cattle, were in quarantine, with 316 sick.

September 20, 1879, 100 herds were in quarantine, of 850 cattle, with 316 sick. Some herds of milk-sick cattle were not allowed to remain in the State, and were sent back to Philadelphia.

September 27, 1879, 101 herds were in quarantine, of 912 cattle, with 366 sick.

October 11, 1879, there were 101 herds, of 888 cattle, in quarantine and 341 sick. In a herd in Ocean County all the infected cases (29) were destroyed.

October 18, 1879, there were in quarantine 97 herds, of 804 cattle, with 308 sick.

November 1, 1879, 98 herds, of 792 cattle, were in quarantine, with 272 sick. Up to this date the State had killed and paid for 37 animals, and owners had either killed or consented to the destruction of 249 in addition.

November 15, 1879, 98 herds, of 782 cattle, with 256 sick, were in quarantine, and inspections were being made in Essex, Monmouth, Hunterdon, Union, Hudson, Morris, Mercer, and Bergen Counties. When slaughtered by the State, prices varied from $6 to $10, the latter generally being the price allowed.

November 29, 1879, 99 herds, of 801 cattle, were in quarantine, with 258 sick. Salem, Gloucester, and Cumberland Counties were included in this inspection.

December 13, 1879, 99 herds, of 865 cattle, with 257 sick, were in quarantine.

January 10, 1880, 97 herds, of 841 cattle, were in quarantine, with 247 sick.

January 26, 1880, 91 herds were in quarantine, with 215 sick.

February 7, 1880, 88 herds, of 822 cattle, were reported in quarantine, with 201 sick.

February 28, 1880, 82 herds, of 728 cattle, were reported in quarantine, with 182 sick. In all there had been killed and paid for by the State 76 cattle in 29 herds, a small compensation being allowed.

After this, until March 10, when the law was repealed, no further record of numbers appears in the letter-file.

This outline of data is valuable as showing the alleged number of cases or of centers of infection, and how defective was the authority given or exercised under the law. As a means of discovering localities of the disease, so far as the diagnosis thereof was correct, valuable information was secured, but the process of stamping out the disease was ineffective. It does not appear that slaughter of cattle was ever made unless by consent of the owner or by agreement with him as to price, in case he sought payment. In those cases the usual price given was $10. It is easy to see that such a law could never have eradicated the disease. It neither attempted to kill all the affected animals nor prevent the spread of the malady by inoculation, but it did no doubt prevent the common mingling of cattle, and so allowed it to die out in some of the herds in which it prevailed. Old cases were not killed as fast as new cases occurred, so that November 1, 1879, up to the time in which the law was vigorously enforced, there were more sick cattle on hand than in the previous months when the machinery of the law was fully in operation and extensive inspections had been made. Notwithstanding the denial of many, it must be admitted that this Bureau revealed considerable prevalence of the disease. The facts that appear as to total losses and slaughter does not seem to point to a malignant type of disease, while the fact that scores of cattle in scores of herds were kept on hand and declared to be sick shows that many get well and that the contagion is not diffusible very far from the center. It also probably indicates that chronic cases get so far well as not to impart the disease. It must be borne in mind that very many of the cases thus carried forward from month to month were cases which had recovered or did recover.

This law was repealed early in March, because its execution had become unpopular in the State, especially by reason of its expense, it having cost about $30,000. At that time it was not supposed any new law would be enacted, but some facts as to acute cases coming to the knowledge of some members of the legislature just at the close of the session, an act was passed directing the State Board of Health to have the matter in charge. The following is the text of the law:

A supplement to an act entitled "An act to establish a State Board of Health," approved March 9, 1877.

1. *Be it enacted by the Senate and General Assembly of the State of New Jersey,* That in addition to the powers conferred by the act to which this is a supplement, said board shall have full power and authority to examine and determine whether pleuro-pneumonia, rinderpest, or any other contagious or infectious disease exists among animals in any county in this State; and that the sum of five hundred dollars is hereby appropriated to defray the actual necessary expenses of said board while making such examinations.

2. *And be it enacted,* That in event of any contagious or infectious disease as aforesaid breaking out or being suspected to exist in any locality in this State, it shall be the duty of all persons owning or having any interest whatever in said cattle immediately to notify the said board of health, or any one of them, of the existence of such disease, and thereupon it shall be the duty of said board of health or any member thereof to immediately proceed to the place or places where said disease is reported to exist, and to quarantine said animal or animals, and take such precautionary measures as shall be deemed necessary; to prescribe such remedies as in their judgment will be conducive to the recovery of such animal or animals, and to enforce such regulations as may be adopted by said board of health.

3. *And be it enacted,* That the board of health aforesaid, and all such assistants as they may appoint, whenever in their judgment or discretion it shall appear in any case that the disease is not likely to yield to any remedial treatment, or whenever it shall seem that the cost or worth of any such remedial treatment shall be greater than the value of any animal or animals so afflicted, or whenever in any case such disease shall threaten its spread to other animals, to cause the same to be immediately slaughtered, and their remains to be buried not less than four feet under ground, and all places in which said animals shall have been kept to be cleansed and disinfected.

4. *And be it enacted,* That in all cases where animals afflicted with, or which shall have been exposed to, shall have been slaughtered or killed by the order of the said board of health, or their assistants, it shall be the duty of said board to appoint three competent and disinterested freeholders to appraise the value of the animals so killed or slaughtered at the time they were so killed, who shall be affirmed or sworn before proceeding to act to make a just and true valuation of said animals so killed at the time of their slaughter, two-thirds of which said valuation or appraisement shall be paid to the owner or owners by the State.

5. *And be it enacted,* That any person or persons refusing or neglecting to notify said board of health, or any of them, of the existence of pleuro-pneumonia, rinderpest, or any other contagious or infectious disease among cattle, shall be deemed and adjudged guilty of a misdemeanor, and upon conviction shall be punished by a fine of not more than two hundred dollars, or by imprisonment not exceeding one year, or both, at the discretion of the court.

6. *And be it enacted,* That all bills for money expended under this act shall be audited by the comptroller of this State and then submitted to the governor for his approval, and after being thus audited and approved by the governor, shall be paid by the state treasurer upon warrant of the comptroller.

7. *And be it enacted,* That said board shall keep a full record of their proceedings and shall publish the same in the annual report of the State board of agriculture, yearly and every year during the existence of this law.

8. *And be it enacted,* That if any person or persons shall knowingly either buy or sell or cause to be bought or sold any animal or animals affected with pleuro-pneumonia, rinderpest, or any other contagious or infectious disease, all such person or persons shall be deemed and adjudged guilty of a misdemeanor, and upon conviction thereof shall be punished by a fine not exceeding two hundred dollars, or imprisonment not exceeding one year, or both, at the discretion of the court.

9. *And be it enacted,* That in case an emergency shall arise and a larger sum shall be deemed necessary than the amount appropriated by the preceding sections of this act, said State board of health shall present the facts in evidence to the president of the State agricultural society, and the president and executive committee of the State board of agriculture, who shall authorize such additional expenditure as in their judgment they may deem the exigency of the occasion to demand: *Provided,* That in no case shall the amount of money thus authorized to be expended exceed the sum of five thousand dollars in any one year.

10. *And be it enacted,* That all acts or parts of acts inconsistent with this act be and the same are hereby repealed, and that this act take effect immediately.

Approved March 12, 1880.

The secretary of the State board at once put himself in communication with the veterinarian in chief of the former bureau, and received a complete list of the herds that at that date (March 10, 1880), were in quarantine, being 110 herds. To these it was at once necessary to attend in order to relieve owners from undue restriction, and also to test the amount of pleuro-pneumonia still remaining in the State. Accordingly the services of three of those who had served under the former bureau were secured, and they were directed at once to visit or communicate with each owner, to examine cattle and to retain in quarantine any found sick. The work was rapidly pushed forward. As a result, it soon became apparent that whatever might have been the former extent of the disease there remained but few evidences of it now. Some chronic cases were reported and a few marked suspicious. One acute case was found in Union County, near Salem. While the disease needed careful watching, we were led to the conclusion that it was not now largely prevalent in the State, and that only a few localities needed any restriction. We quote as follows for 1880, from the report as made to the legislature of New Jersey:

In April, 1880, cases of pleuro-pneumonia were found to exist at Manasquan, Monmouth County, a chronic case in Bergen County, one acute case and five chronic cases near Lyons Farms. Inspection was also made of a chronic case and one suspected acute case in Hudson County, but, after two or three examinations and personal visits, the cases were found to be of long standing and believed to have passed the infective stage. In May the disease showed itself at Forked River, but disinfection was at once resorted to. One cow died, and the others did not contract the disease. Both these, and the cases of the month of April, besides some chronic cases previous to these, were isolated and kept under watch, but in no cases did the disease extend.

Early in June we received notice of the disease as appearing in the vicinity of Union County. It was confined to one herd of about twenty cows, three of which died, and two were slaughtered under our direction. Four chronic cases were isolated and carefully watched. Inoculation had been resorted to for this herd after the first cases. We shall have occasion in another connection to present the facts in evidence and to discuss inoculation in reference thereto.

Early in July pleuro-pneumonia was found to exist near Mount Holly, Burlington County, in an infectious form, and measures were taken to prevent its spread. Dr. C. K. Dyer was appointed as an additional inspector. Two cattle sick in the herd of Mr. Deacon were slaughtered, and a neighbor's herd was kept under isolation on account of suspicion of disease which had caused some deaths, and in reference to which several veterinarians differed. The cases in Burlington County were traced to their source, and precautions used to prevent extension. They had been traced to purchases made in the Philadelphia stock-yards.

By this time, in the re-examination of over 100 herds found in quarantine at date of March 10, in the examination of reported cases, and in the oversight and disposition of actual cases of the disease, the $500 appropriated in the first section of the bill had been expended. As the disease was known to exist at a few localities and to need continual supervision, and as there was every reason to fear still an occasional outbreak, the State board of health presented the facts to the president of the State agricultural society and the president and executive committee of the State board of agriculture, as provided in section 9 of the bill of March 12, 1880. At a conference had at Newark, the board was authorized to expend $500 additional, if need be, and if more was required, again to present the facts in evidence.

In the latter part of July and early in August there was an extension of pleuro-pneumonia in townships a few miles about Mount Holly, and notices of suspicion of it in other places. Several cattle were sick in the vicinity of Freehold, but the disease did not prove to be pleuro-pneumonia. Edward J. Davis, of Clayton, Gloucester County, notified us of it in his herd. S. W. Steelman's herd was also suspected. Two animals were affected with pneumonia, but we were not able to identify it as of the contagious variety. Mr. George Borton, of Burlington County, whose herd was quarantined last year, also reported a suspected case, which proved to be laryngitis. About the first week in August, notices reached us that two or three more herds were affected in the vicinity of Masonville, Burlington County. Examination showed the disease on the farms of Mr. Joseph Killer, Mr. H. P. Troth, Mr. Daniel Emly, and Mr. Job H. Gaskill, all within a few miles of Mount Holly. The third cow of Mr. Deacon also needed to be slaughtered.

The disease manifested itself at these several points in Burlington County, not in communication with each other. We found it necessary to order the slaughter of eleven cattle in five different herds, and to take active precautions to avoid the extension of the disease. When possible, we called on one or more members of the township board of health or township committee as examiners, and also notified these boards for the purpose of securing closer local inquiry and oversight. We at once slaughtered all cases of the disease. Farmers and stock raisers in this whole section realized the threatening danger of the malady and co-operated with us in every way. Most of these cases, and probably all, could be traced to cattle sold by one dealer to these various persons. Out of his herd we found that he had lost and buried two cattle, from some disease unknown to him, before he had sold the others.

Soon after we were notified of an outbreak of the disease and the loss of some cattle near Hackensack, Bergen County. The cases were obscure, but careful investigation showed them not to be contagious pleuro-pneumonia.

A notification from Camden County, about 3 miles from Camden, proved more serious. A Philadelphia dealer had sent three cattle with calves, for sale, to a farmer. About five weeks after two of the calves died and one cow with symptoms recognized before death, and proved by examination after, to have been pleuro-pneumonia. The other cow had the disease, and due notice was given to the Pennsylvania owner. Another cow had been sold to a dairyman 3 miles distant. We at once sought out this animal and found her with premonitory symptoms, and secured thorough isolation. These cases illustrated a long incubative stage, and also how calves may become diseased and be the most rapid victims. These animals were afterwards found to have come from the same herd which had infected Burlington County.

Up to the date of September 1 all of these cattle had been killed by order of this board. There were other cases which died or could be so isolated as not to require slaughter. Many cattle we had occasion to see died from other causes, and the suspicions as to pleuro-pneumonia were not sustained. The cases in Camden County were not disposed of because they could be isolated, and also because the owner, living out of the State and having brought the cattle into the State, was held responsible to dispose of the same. All the other cases of slaughter up to this time, except the two in Union, Union County, were in the townships about Mount Holly, Burlington County. The price paid was generally $20.

Monthly notices were exchanged with the Pennsylvania authorities, and their attention was drawn to the infected Camden County cattle, which had been sent over from Philadelphia for pasturage. We procured from the United States Government copies of its excellent monograph on the diseases of cattle and distributed them.

In September pleuro-pneumonia continued to demand our careful attention in Burlington County, and it was found necessary to slaughter several more cattle, and some others died. Six different herds in all had been affected, and others had been examined in which cattle were lost from other causes.

On September 11 a conference was held with some of the local boards of health, that they might aid us in limiting the contagion, and also to settle the grounds upon which valuation should be made. Information received as to the cases in Camden County showed more fully that these had been derived from the one herd which had occasioned it in Burlington County, and which had received it from Philadelphia.

In a single herd in Bergen County, one case occurred where it had been the previous year, but there has been no extension.

The report exchanged with Pennsylvania, October 1, showed no actual case of pleuro-pneumonia in the State known to the authorities, although two or three herds were still in quarantine. The Philadelphia stock-yards were being carefully guarded.

In October new cases occurred in some of the herds already affected, but in most it seemed subsiding. Two new cases occurred on a farm adjoining one in quarantine, the cattle having been together previously. New cases were reported in Passaic County which required examination, but proved to be another malady.

In November one case was reported in a new herd in Burlington County, but in all but one herd, that of the farms quarantined, there were no new cases of the disease.

In Camden County a case occurred in the former herd. Even where a herd is exposed all do not take the disease. Isolation and disinfection will accomplish much. The complete success which followed as to the eradication of the disease in Burlington County was most satisfactory. Some cases from Mercer County required investigation, but proved not to be the disease. One farmer in Camden County also lost 9 cattle from an obscure form of disease, but there were no lung lesions.

In December a new point of infection occurred in Camden County through cattle bought at the Pennsylvania stock-yards, which had come from Virginia.

The disease also existed on three farms in Salem County where it had previously occurred, but was distinctly traceable to some cattle purchased from a drove brought from Philadelphia. Every case that occurred this year in the State was traceable to animals recently purchased. In most cases the infection has been derived from one place in Burlington County, or from cattle that came from some State south of us. We know of one sale of cows that cost the State about $1,000.

It is in vain to battle with such a disease by half measures. The law, it was decided, did not give authority to inspect cattle except upon the application of owners. Hence, in cases of droves brought into the State we were left defenseless. It was only by close watchfulness and the co-operation of local boards of health and of individual farmers that we were able to keep the disease in check. In no case did the disease spread from one herd to another so far as we know, after it had come under the jurisdiction of our board. Several thousand circulars were distributed at small expense, and where cases of this or other contagious diseases occurred we sent, through books and pamphlets, other information. Every local health board in the State had such information as to be in communication with us, and so apprised us of the evil. When the disease occurred we found the people generally disposed to favor active measures.

Early in January, 1881, a case was reported in Mercer County near Princeton. The animal was early separated by the owner from the rest of the herd. He had had pleuro-pneumonia in his herd two years before, when living on a different farm, although this case had no connection therewith.

One of our veterinary inspectors saw the animal, and the owner finding the disease to be pleuro-pneumonia did not wait for appraisement but at once destroyed it, and the post-mortem showed the usual lesions.

Communication, as usual, was made to the Pennsylvania authorities in the form of a monthly report. Report was also made to us as to pleuro-pneumonia on the Pennsylvania side of the Delaware, and of the ill condition of store cattle sent to this State.

Reports from the veterinarian in February revealed no new cases of the disease.

In March the death of three cattle from supposed pleuro-pneumonia was reported to us from Sussex County, but the facts in evidence showed it not to be this disease. The investigation of suspected cases and decisions as to those which require and those which do not require investigation often claimed the attention of this board and its assistants. It is evident that those not having seen much of the disease not infrequently err in diagnosis.

A change made in the law by the legislature made it more effective. Soon after the passage of the supplement, an accompanying circular, with the law, was sent to every local board and to all the various organizations which the board of agriculture represents. The board of health, in its oversight of the contagious diseases of animals, has sought to spread broadcast through the State such information as shall lead all owners of stocks to guard against diseases which are communicable, and so to interest the local health boards that early information may be sent to us and early precautions be taken. The eight circulars as to contagious diseases of animals have had a wide circulation.

The board of health of Little Egg Harbor Township, Atlantic County, had its attention called to a fatal cattle disease in their section, and reported the facts to us. We were able to satisfy ourselves and them that the disease was not pleuro-pneumonia.

During the month other cases were reported to us in Hudson County, but our inspector there, after diligent examination, satisfied himself that the reports were incorrect.

In order to be able more fully to certify as to Hudson County, we had a correspondence with its board of health, and sought such an arrangement as would subject all cattle within its limits to inspection and enable our local inspector to become fully acquainted with any possible places where cattle were kept so as to be subject to disease. By this arrangement we sought still more thoroughly to guard against contagious diseases. We regret to say that the cities of Hudson County have many breeding places for cattle diseases, and that the sanitary care of animals is overlooked.

During April some correspondence was had with the Department of Agriculture at Washington as to some authorization it had given as to inquiring into pleuro-pneumonia in this State in behalf of the General Government.

Early in May information was received that a cow belonging to L. G. Howell, of Millstone, Middlesex County, had pleuro-pneumonia. The cow soon died and a veterinarian was sent to examine and, if need be, quarantine the rest of the herd. He was also sent to see the person at South River from whom the cow had been purchased, and found one of his cattle ailing. This was also separated, and a subsequent visit confirmed the suspicion of pleuro-pneumonia. The animal was at once appraised and killed. These cattle had been purchased at Staten Island. About the same time a case of the disease was found at Black Horse Tavern, not far from Dean's Station, Middlesex County. The animal died and proper regulations and disinfectants were at once adopted.

The center of infection which had been established near South River required careful oversight, because of the number of cattle in the roads and on a river meadow. It was necessary to slaughter more animals and to notify owners of their risks and to warn against Staten Island cattle.

It was found that some cattle near South Amboy were diseased, and that it was necessary to guard against infection at that point. We had new evidence that persons who buy cattle from other States should be required either in some form to register or that their names and addresses should be kept and their reliability investigated. The disease reaches our State chiefly through those who have bought infected herds at nominal prices and then sell those which have not as yet shown the disease, but it can be stopped by holding the dealers responsible. Even when they do not know the animal to be infected, many of them do know that they never could have purchased the stock so cheaply unless some concealed reason for the sale had existed. We were afterwards able to trace all these cases to a Staten Island butcher who had ferried the cattle over to New Jersey and sold them.

Early in July two cases of pleuro-pneumonia were discovered at Camden. One cow died and the other was slaughtered. These, as have some other cases, showed us that the disease was prevalent at some points to the south of New Jersey, and that constant vigilance is required to prevent the spread of the malady by means of cattle brought into the State. Inspection of herds may detect those actually sick, but it cannot detect what animals have been exposed to infected herds. It is therefore necessary to be on the watch for cases that actually occur and to deal with them promptly, so as to prevent an extension of the contagion. An examination of other cattle showed at least twenty as already more or less diseased or as suspicious in extending contagion. It was therefore the judgment of those acting that the safety of the county and of the State demanded the condemnation and slaughter of all of this dangerous herd. They were accordingly condemned and appraised under the law.

Near the close of the year report was made to us that contagious pleuro-pneumonia had found its way into a herd of one hundred cattle near Piné Brook, Morris County. We found not only that this was the case, but that thirteen had died, and that the owner, not knowing the State law on the subject, had caused all the rest to be inoculated. The herd was found in such a condition that seven were at once killed and the rest quarantined. Of those inoculated, less than one-half had been protected. The law very properly prohibits inoculation, except by permission of the board of health. The owner of the cattle had not intended to disobey the law of the State, and the inoculator also claims ignorance. We satisfied ourselves that cases now occurring had not been caused by the inoculation several weeks before. The owner consented to the killing of one without appraisement, in order to test the distinct lesions of inoculation.

Much has been saved to the State by preventing the disease from spreading. It is a still higher and more important work to prevent it from occurring at all. This is to be done by giving such authority over cattle brought into the State as shall secure all and every precaution consistent with a not undue interference with transportation.

The spreading of disease, after it is introduced, is prevented in two ways: by giving plenary authority to the proper officers and inflicting penalties for non-report, and by spreading such information among farmers and stock-dealers as shall lead them to keep new purchases separate, or to take the earliest precautions in case of sickness. This board, with moderate expense, has been able by circulars and by gifts sought and received from the United States Agricultural Department to place in the hands of local boards of health much information on these matters. A recognition of the possibilities of disease and the precautions to be used enables owners to guard against epidemics.

The following is the record of locality as to actual cases and the disposition made of them:

REPORT OF W. B. E. MILLER, D. V. S.

December 2, 1880.—Visited farm of T. Hilton, Wrightville, Camden County, and December 3 revisited same farm and slaughtered four cattle at the expense of the State; balance sold to butchers or killed at the expense of owner.

December 7.—Found pleuro-pneumonia on Riley's farm and evidences of the same on Steward's; ordered diseased stock to be isolated and killed and the exposed to be quarantined, fattened, and slaughtered as rapidly as possible, all of which was carried out to the letter.

December 9.—Visited Kirkwood, Camden County; examined some steers belonging to Ephraim Tomlinson; found one in fatal stage of contagious pleuro-pneumonia; advised immediate slaughter at owner's expense, isolation of exposed animals, disinfection of premises, and sale of other stock to butcher.

December 13.—Revisited E. Tomlinson's farm, accompanied by Dr. T. B. Rogers, examined the entire herd of seventy steers, found several slightly affected, advised isolation and slaughter at the very earliest date.

January 13, 1881.—Visited farm of William Y. Johnson, Princeton, Mercer County; examined his entire herd of cattle; found one isolated cow in last stage of pleuro-pneumonia, slaughtered her at owner's expense, and made *post mortem* to confirm diagnosis. No further cases.

May 5.—Visited farm of William Sears, near Dean's Station, Middlesex County; found carcass of cow that had just died from pleuro-pneumonia; made a *post-mortem* examination thereon that showed the disease.

June 15.—Revisited same farm; found no further evidence of any disease.

June 18.—Visited South River, Middlesex County; examined herd of Robert L. Armstrong and others; found three sick with contagious pleuro-pneumonia; condemned, appraised, slaughtered, and made *post-mortem* on their carcasses, then buried the same. Dr. McLaughlin had condemned some also.

July 1.—Visited stable of James Breen, of Camden; found cow in last stage of pleuro-pneumonia. She died the following day, when I saw and examined her lungs, both of which were thoroughly diseased.

July 7.—Revisited South River. Two of Armstrong's herd had died since last visitation; five more were found sick; were appraised, slaughtered, and disposed of as before.

July 8.—Re-examined the balance of the herd and advised the owner to slaughter the entire lot. The herd belonged to several persons, who pastured along an open salt marsh.

July 15.—Appraised the value of cow condemned at ferry inspection a few days before and since quarantined at my own stable. She was owned by Mr. Caleb Ridgway, of Vincentown, he having purchased her at the West Philadelphia stock-yards, and was removing her to Vincentown, where she was found to be diseased; slaughtered and made a *post-mortem* examination.

July 21.—Visited South River, Middlesex County. Re-examined herd of R. L. Armstrong and others, and resolved to dispose of entire herd. There were 20 cattle, belonging to several owners.

July 28.—Attended slaughter of said animals, superintending the same, designating such as were fitted for food and consigning others to the offal-houses.

December 7.—Examined one lot of six cows coming from Mount Holly, and another of five coming from Elmer, Salem County, in each of which was one cow that I detected as having lung trouble, in my ferry examinations. Ascertaining that they were going direct to the slaughter-house, I allowed them to pass on and went to witness their slaughter.

REPORT OF JOHN A. M'LAUGHLIN, JR., D. V. S.

May 9.—Visited Little Washington; found pleuro-pneumonia *contagiosa*. Quarantined place.

May 10.—Visited East Millstone; examined herd where pleuro-pneumonia had been; separated two suspicious cases; quarantined place.

May 12.—Visited Little Washington; killed one cow suffering from pleuro-pneumonia *contagiosa*.

December 9.—Went to Pine Brook; diagnosed pleuro-pneumonia; isolated and quarantined.

December 11.—Revisited Pine Brook; killed seven cattle.

December 20.—Went to Pine Brook in company with Dr. Hunt; killed one cow. The herd was inoculated.

It was our habit, nearly always, to have careful *post-mortem* examination made; not because this was necessary to the diagnosis of the disease, but because it seemed more fully to show the various conditions in which the lungs are found. During all the spring and summer, a veterinarian, employed on a salary by the General Government, was engaged in examining into the disease in this State, independent of this board. He had claimed the disease to exist to a greater extent than was known to this board, and that he himself was aware of it. This appointment made it incumbent upon him to furnish all evidence to the General Government. The first case reported bears date March 25, 1881, and the last, September 16. It was found that it was mostly a selection from a record of cases in a previous year. On the list, besides the South River and the Millstone cases already under care of this board, but four acute cases are recorded. We corresponded with parties named and were unable to find evidence of the existence of the disease in any herds not then under our oversight. The record served to show how thoroughly the board had been apprised of the disease.

We adopted the plan of non-concealment of the disease, except that when requested by the owner no mention was made of the presence of the plague in the herd until after the quarantine had ended.

From this time on, the cases of pleuro-pneumonia, as they occurred after December, 1881, are to be found in the reports of the State board of agriculture of New Jersey. In West Jersey, where the disease was at first very troublesome, the constant watchfulness of the veterinarians, with the aid of local authorities, have so far succeeded in preventing any spread of the contagion. Sporadic cases have occurred, which were generally introduced from the Philadelphia stock-yards. Farmers generally are apprised of the danger, and the disease is not allowed to become epidemic. In the year 1882 our chief trouble was in Essex, Unoin, and Hunterdon Counties. Several herds in the vicinity of Newark were affected. Besides slaughter, it was found necessary frequently to resort to inoculation in diseased herds. A few herds also in the vicinity of White House, Hunterdon County, were affected. In these, resort had been had to inoculation, without the knowledge of the board. Owing to several circumstances, the disease was not as readily checked as it might have been. In Union County, several herds near Elizabeth were affected and some were inoculated.

The following is a statement of J. W. Hawk, D. V. S., as to inoculation in Essex and Union Counties:

> Since my appointment by the State board of health, I have inoculated seven herds. In one herd of twenty-four cows belonging to ——— ———, East Orange, I killed three cows and inoculated the rest of the herd. After inoculation, I took the temperature of some of the cows, and found it as high as 106⅓° Fahr. in some cases.
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> In a herd of twenty-three cows at Waverly I killed three and inoculated the others at the owner's expense. To satisfy myself if there were any probability of the cows taking contagious pleuro-pneumonia after inoculation, I placed acute cases among the rest of the herd, but none took the disease. I was sure they would not. Temperature of one of the cows killed was 105¾° Fahr.
>
> The first herd I inoculated February, 1 83. It belonged to Mrs. Mary Pfeifer, and contained fourteen cows. I killed three cows; temperature, about 105¾° Fahr.
>
> I inoculated Mr. Henry Hanfield's herd of sixteen cows. Cow killed; temperature, 104⅓° Fahr.
>
> Mr. Isaac Heddin's seven cows I inoculated; temperature of one killed, 105° Fahr.
>
> In the other two cases where I inoculated the cattle did well. In every herd that I have referred to not one cow took sick after inoculation. They are in better health than before inoculation. Of course, while the inoculation was taking, there was a gradual falling off in the quantity of milk from two to three quarts; but since the inoculation they are in better condition and give more milk.

In all about 115 cattle have been paid for by the State. About six have been killed without pay in cases where inoculation had been done by owners without the knowledge of the board. Several were killed by owners without asking compensation. The whole number killed has been about 175. The disease has been found in about 75 herds.

During the year 1884 the disease has prevailed in several herds, but we have now been able to release all herds from quarantine except several in Hudson County. We are now engaged in attending to them, and hope, as far as possible, to eradicate the plague in that county.

These statements of the disease, as met with and treated in the State of New Jersey, are the nearest exhibit of the disease as it has existed. While it is not claimed that every case has been discovered, it is believed, that with the facilities for finding out its existence, and for the most part a favorable feeling towards the law, except on the part of dealers in poor cattle, and cow keepers in the city stables, nearly all the cases have come to the knowledge of the State board of health.

It has been interesting to watch how unreliable has been public rumor as to the disease. Sometimes two or three cases would, through some rumor or the officiousness of one or more parties, get into the papers, and be sounded through the land. At other times, when the disease was much more prevalent, the news would extend but little beyond the adjoining farm. In November, 1881, when the governor of Illinois quarantined against fifteen counties in the State of New Jersey, the disease did not and had not for months existed in over three of the counties named. Neither himself nor his informants had asked for or obtained any information from the board in charge of contagious diseases of animals in the State, or from any of the State authorities.

The experience of the board during a period now extended over nearly five years leads it to believe that it is or has been fully practicable to stamp pleuro-pneumonia out of this country. We would not to-day have a single case or a single infected center in the State if only the General Government had aided in controlling the transportation of infected cattle. It is because of defects in inter-State guard that it invades us. It is not a disease like the horse epizootic or influenza, which is easily carried in the air through long distances. The destruction of the contagion and its failure, in so many instances, to spread extensively from infected centers, plainly show that the contagious particles are not of as persistent vitality as those of some other diseases. While most authorities claim that inoculation should be relied upon as a general protection, it does come to our aid where large herds are exposed to the malady. With all the disadvantages of position as related to transportation, and with the fact that in these years we have had to deal with severe outbreaks in bad localities, we have at least succeeded in preventing a widespread seizure. We expect still to be able to do this, but we shall never be able fully to eradicate the disease. It will over and over be imported to us from adjoining States. It will be a great gain when in all cities the board of health shall be able to require the registry of all places where cattle are kept and a quarterly report from the inspectors of health as to their condition. If there could be added a system of abattoirs by which all meat that is killed could be liable to inspection, it would not only protect the public from meat unfit for use, but would greatly aid in the eradication of animal diseases.

What shall be the future of this country as to the prevalence of pleuro-pneumonia will be decided by the efficiency with which the General Government provides for its extinction.

ERGOTISM AMONG CATTLE IN KANSAS.

Hon. GEORGE B. LORING,
Commissioner of Agriculture:

SIR: In obedience to your telegram of the 5th of March, requesting me to go to Neosho Falls, Kans., and there to investigate a disease among cattle, I forthwith prepared myself and left on the 6th, arriving at Neosho Falls in the afternoon of the 8th. On my way to Neosho Falls I was delayed overnight at Burlington, Kans. On the same evening I read a report in the Kansas City *Journal* of a meeting which had been held at Neosho Falls on the evening of the 6th attended by his excellency Governor G. W. Glick, Lieut.-Gov. D. W. Finney, who, in company with other State officials, Dr. A. A. Holcombe, D. V. S., of Leavenworth, Dr. Wilhite, of Emporia, and a number of stockmen, had by special train proceeded to Neosho Falls, and investigated the disease among cattle in Woodson and Coffey Counties, and upon the authority of Drs. Holcombe and Wilhite said disease was pronounced to be the contagious foot-and-mouth disease. At the meeting in the evening a quarantine committee was appointed, with Lieutenant-Governor Finney as the chairman. This committee was instructed to quarantine all infected cattle and premises, to guard against further diffusion of the dread disease. Upon reading this article I was greatly surprised and alarmed. I had no reason to doubt the correctness of the diagnosis by Dr. Holcombe, whom I knew from personal knowledge to be a very competent veterinarian.

On my arrival at Neosho Falls, Woodson County, late in the afternoon of the 8th, I was met by Lieutenant-Governor Finney and Dr. Holcombe, who immediately procured a conveyance and accompanied me to the residence and farm of Mr. Daniel Keith, situated in the southeast corner of Coffey County, 5 miles north of Neosho Falls. Here I was conducted to a small pen or inclosure which contained 12 head of yearling calves. This pen measured about 40 by 60 feet, and was inclosed on two sides by a rail fence, by a hay rack and corn crib on the west side, and horse stable and corn crib on the east. The pen was well bedded with hay and straw. On entering the inclosure I walked along the hay rack and gave the hay a hasty examination, but found it clean, bright, and sweet, it being wild hay made on bottom lands. I also looked for ergot among the grasses which composed the hay, but discovered only two or three heads of wild rye which were ergotized; the amount being insignificant I gave it no further thought at the time. My expectation, after reading a description of the symptoms of the disease among the cattle, was to discover ergotism, should the disease prove not to be the genuine foot-and-mouth affection. I inquired if any of the pregnant cows and heifers had aborted, and was told they had not; but my attention was called to one white cow, six years of age, which presented many ulcers on the udder and teats. These ulcers, according to the description of Mr. Keith, first appeared as vesicles or blisters about three weeks previous, but at the time I saw her they were covered with scabs, and were healing off. Mr. Keith stated that this cow was suckling a ten day's old calf when she first evinced symptoms of the disease, and that two days thereafter the calf died, having succumbed to a severe diarrhea.

The yearlings in the pen presented the following conditions: Four of them had lost both hind feet, the separation taking place at the fetlock joints; 5 had each lost one hind foot; and 3 were about to lose both hind feet. The latter cases presented a well-defined line of demarkation at or above the fetlock joint, extending and encircling the limb in a straight or slightly oblique line; the upper parts of said line presented a healthy surface, discharging a small amount of laudable pus, and were healing under a scab; the limbs below this line were perfectly devitalized and shrunken in a dry gangrenous condition. In the clefts of several of the feet I found considerable evidence of previous ulceration, and loosening of the walls of the foot around the bulb of the heel. Those animals which had lost their feet were apparently in a healthy condition, a slight elevation of temperature being the only appreciable evidence of functional derangement remaining, aside from the crippled limbs. They ate well; licked themselves; the stumps were scabbing over and healing; they occasionally got up and hobbled along for a few steps, then dropped down.

I next examined their mouths, which revealed denudation of the mucous membrane and discolored patches on the nose, lips, tongue, and roof and elastic pad. In some of them only two or three of these discolored spots would be found on the lips, pad, or the roof of the mouth. The discolored or denuded patches did not extend deeper than the submucous connective tissue, and presented a brownish yellow or rusty color; they were irregular in outline, and of various dimensions, from the size of a pencil's point to half an inch in diameter, and were not circumscribed by any reddish or inflammatory border.

No soreness or inconvenience was manifested in the act of eating. Temperatures ranged from 102° to 103.8° F. I saw a number of other cattle in various stages of lameness, also some which had recovered from lameness, yet no very recent cases were reported by Mr. Keith, from whom I received the following brief history:

He first became aware of the trouble among his cattle on or about the 23d or 25th of December last, when he noticed a peculiar jerking up of the hind legs among a number of the calves. They would first jerk up one foot and then the other, or shake the foot as if they wanted to shake off a foreign body, and acted as if they could not place the affected foot to the ground. They would then hobble along a few steps, and walk off moderately well or lie down. When they stood quiet they arched the back and dropped the head.

Some of them slobbered or frothed at the mouth, and would not eat hay very well. In the course of two or three days they persisted in lying down nearly all the time; swelling about the coronet then became apparent, extending as high up as the fetlock, or even higher in some cases, which was attended by great heat and tenderness.

Soon after this swelling appeared—a very few days—a band around the leg would then appear, the skin becoming contracted, dry, and hard; next the skin broke and a sore made its appearance. This sore encircled the leg and gradually deepened until complete separation of the limb at one of the joints occurred. The time consumed from the first appearance of the disease until the final dropping off of the dead portion of the limb would be from three to four weeks. In some cases he noticed soreness and ulceration in the clefts between the claws. When he first noticed the jerking up of the feet and limbs he thought it was due to impaction of mud between the claws; therefore he examined some of the feet, but found no impactions. On the 1st of January about 30 head of the calves manifested lameness. All of these calves were fed upon wild hay and shelled corn during the fall and winter. All the medical treatment which these cattle received was one application of muriatic acid around the limb where the line of soreness existed. The described 12 head of yearling calves, and 51 more of the same age, he bought from Mr. J. Davis on the 11th of December, and took them home on the 12th. Mr. Davis had bought these calves from different parties within a radius of 10 miles south and east of Neosho Falls.

Leaving Mr. Keith's place, we went to the farm owned by Mr. A. C. Goodrich, which is occupied by Mr. Edward Hindman, who is the overseer of the stock on the farm. The Goodrich farm is divided from the Keith farm by a public road running north and south. On the Goodrich farm we saw 20 head of cattle, all of which were two years old and upwards, which had then lost or would eventually lose one or more feet, or parts of them, and 2 of them were about to lose all their feet.

In one of these cases the line of demarkation was 6 inches above the fetlock, and in the other 4 inches, while some of them had lost only one claw or one foot at the second joint. All of these cattle presented greater or less discolorations, erosions, or ulcerations on the lips, tongue, or roof of the mouth, and in several the mouth lesions were much more prominent than in any of the Keith cattle. A two-year old red and white steer, which had lost both hind feet at the fetlock joints, presented, upon examination of the mouth, brownish-yellow patches on the roof, covering two-thirds of the space between the pad and soft palate. These patches coalesced and were elevated one-sixteenth of an inch, possessed a well-defined border and a flattened surface. On the pad were two large oval or oblong ulcers, one of which measured an inch and a quarter in length. On the gums of the lower jaw, inside of the lateral and corner-incisor teeth of the right side, appeared one large ulcer, which possessed a more reddish and inflammatory appearance than those on the pad. On the right side of the thick portion of the tongue existed an ulcer which had an excavated bottom and a greasy and dirty-looking appearance; this was 1½ inches in length and of an oval shape. Temperature 103.8° F. A two-year-old red heifer, which had lost one hind foot at the fetlock joint, and the other was in process of separation at the same place, presented a mouth with lesions exactly similar to the first one, only that they were confined solely to the roof of the mouth.

A third case—a five-year-old cow which was losing both hind feet at the fetlocks—presented discolored patches and small erosions on the tongue, lips, and roof of the mouth, also ecchymosed spots appeared on the mucous lining of the vulva, and mucus mixed with pus flowed from the vagina. An ulcer the size of a silver dime appeared inside of the sphincter ani. Temperature 104° F. Mr. Goodrich, among his herd of 95 animals, had 21 cows and a number of heifers which were pregnant, but none of them aborted.

In an adjoining lot I saw a number of young calves; upon inquiry I received the response that these calves were all well, and had not been with the diseased cattle. A number of hogs also were inclosed in another lot adjoining that of the diseased cattle, and I was told that they had not been in contact with the cattle. Night was now approaching, and owing to the number of people which had gathered, and the excitement which prevailed, it was impossible to obtain any definite or extended history of the outbreak, manner of feeding, surroundings, &c. Therefore I deemed it best to return to town and compare notes with Dr. Holcombe, and return on the next day to make a more thorough investigation, and to obtain a full history. I was now told by some of the stockmen present that they desired to have my opinion on the nature of the disease; that they intended to have the cattle appraised, killed, and buried on the next day, if my diagnosis corresponded with that of Dr. Holcombe. Two of these parties came from Emporia for this expressed purpose.

This placed me in a very delicate and unpleasant position, as I had not given the matter the thorough investigation that I desired to do before making my diagnosis. I had to rely almost solely upon a history as given to me by parties upon whom I could not place absolute reliance; nevertheless I felt compelled to make a diagnosis under these pressing circumstances. Taking, therefore, into consideration, 1st. The history which was given to me by Mr. Keith, which so closely described the first symptoms of the foot-and-mouth disease; the ulcers and previous vesication on the udder of the Keith cow, and the death of her calf soon after the disease manifested itself in the mother; the absence of abortions or the supervention of nervous affections which I expected to find in ergotism. 2d. Attributing the discolorations of the mouth to the remains of previous vesications; the losing of the feet as a sequelæ to foot-and mouth disease aggravated by neglect, and exposure to intense cold. 3d. Accepting the history of a case described to me by Dr. Holcombe, where he discovered an animal on the Keith place in the second or vesicular stage of foot-and-mouth disease in which he found several distinct characteristic vesicles in the mouth, accompanied by salivation, and another vesicle in the cleft of the foot near the heel the size of a silver dime, and which he caused to rupture by a pressure with his finger, and registering a temperature of 104.4° F. 4th. Accepting as true the positive statement of Mr. J. W. Beard, who lives 2 miles south of Mr. Keith. He stated that he had exchanged cows with Mr. Keith on the 18th of February, and that two days after he brought the cow home from the Keith place one of his cows contracted the disease, and that several others followed in quick succession; (this was strong evidence to prove the contagious character of the malady); in the absence of recent cases, placing reliance upon the statement made by Dr. Holcombe, and upon his diagnosis, having no valid objections to offer to it, I was led to concur with him, and announced my belief that the disease among the cattle on the Keith and on the Goodrich farms was epizootic aphthæ. I went out to the Keith farm again on Sunday, the 9th, with the intention of obtaining a complete history of the outbreak, to examine the quality of the feed, water, and soil, to make a more extended examination of all of the diseased animals, and to make *post-mortem* examinations if opportunity afforded it. But when I arrived at the Keith place a stream of people were passing in and out among the cattle. A delegation of stockmen were there with the avowed purpose of paying for and disposing of the diseased animals, and another number of persons were holding a consultation in relation to petitioning Governor Glick, requesting him to convene the State legislature for the purpose of enacting laws and to make provision for the stamping out of the disease. These parties were monopolizing the time of the owners of the cattle, consequently I had to wait for a more favorable day to accomplish my work. This day, however, I made a more extended observation of the cattle on both farms, as I was exceedingly anxious to find a recent case. My search was rewarded by finding a red yearling steer at Keith's, which presented a blister at the anterior border of the soft palate; it was about the size of a silver dime, and had a thin raised pellicle of mucous membrane which ruptured when I touched it. Two small pointed vesicles appeared on the upper surface of the tongue. No excessive salivation was present. A pinkish color was diffused over the membrane of the mouth and tongue. Breathing accelerated; temperature 104.5°. In withdrawing the instrument from the rectum an ulcer was exposed to view, which bled slightly. (On the following day I saw this animal again and found the conditions unchanged, except that the blisters in the mouth had assumed the characteristic brownish-yellow color, similar to those found in the mouths of the worst cases. In the course of ten days this animal had about recovered.) On this day I noticed several animals frothing at the mouth, although they showed but slight indications of lameness, but for want of proper assistance was unable to catch them for closer inspection. On the Goodrich farm I found a number of cows and heifers to have discharges from the vagina, accompanied by thickening ecchymosis, or ulcerations of the mucous membrane of the vaginal walls. I noticed also small blood-clots and mucus on recently-dropped *feces*.

On Monday, the 10th, I looked again for recent cases, but did not succeed in finding any.

Mr. Hindman gave me the following history: He has lived eight years on this place, and has been engaged in raising and feeding stock during that time. He never before had any disease among his cattle. He took into a herd 5 miles east, which was in charge of George Grant, 72 head of cattle last spring, and on the 10th of October brought home 78 head. During the summer 2 died and 5 were sold. Since the return of the 78 head 8 calves have been born. These cattle, since the 10th of October, have been kept in a feed-lot, sheltered by timber, south and east of the house. They derived their drinking water from a pond (surface water) located in a field 20 rods north of the house, and at about the same distance from the residence and yards of Mr. Keith. On New Year's week he took all the cattle, except a few cows and a bull, out of the feed-lots, driving them through a gate south of the house, and then drove them down the public road a distance of 40 rods and turned them into a stalk-field and meadow, from whence they could again return through a gap in the hedge to the old feed-lot and drinking place. He kept 3 cows and a bull in a small field north of the house, from which they went to the same pond for water as did the other cattle. On or about the 10th of January one of these milch cows became lame in one hind foot, and was yet lame when I saw her, but manifested no indications of losing any part of her foot. The next cases of lameness appeared on the 14th or 15th of February, the day after a heavy rain and sleet storm. On that morning a number of cattle were lame, and new cases appeared daily for a number of days thereafter. The lameness attacked cattle regardless of age, sex, or condition, old as well as young, and just as severely. As soon as they became too lame to get around to feed and water they were driven into a small yard on the east side of the house, where they had a covered shed for shelter, and feed and water was carried to them. In this shed the worst cases were kept, and in the west end of it a small stall was partitioned off by a few fence boards, in which the bull was tied. This bull never manifested any symptoms of the disease. This is substantially the history which I obtained from Mr. Hindman on this day. With regard to the kind of feed which the cattle received, Mr. Hindman told me that all the cattle one year old and upwards were fed upon hay made on the bottom lands, that they depastured 30 acres of corn stalks, and lately received wheat straw in addition to the hay. The milch cows, which were kept north of the house, the bull, and the young calves received corn in addition to hay.

On a subsequent visit I was informed, either by Mr. Hindman or one of his hired men, that the hogs had been allowed to remain in the yard with the diseased cattle until they began to gnaw at their dead feet, in consequence of which they were turned into the orchard. I also discovered that the young calves had remained in this yard until they were crowded out by the rapid increase of invalids.

History.—On the 11th of December Daniel Keith bought 63 head of yearling calves from Mr. Joseph Davis, and brought them home on the 12th. Four days previous to this time he had bought 5 yearlings from Nelson Stride, 2 miles south. He bought one from William Inge, 2 miles southeast, about a week later. Bought one from Balt. King, 2½ miles southwest of Neosho Falls, a day or two after the Inge calf. Bought one 4 miles south of Neosho Falls on or about the 10th of February, and on the 20th bought 6 head from Alex. Linn, 1 mile down the river from Neosho Falls. On or about the 23d of December he first noticed the lameness to exist among the calves which he had bought from Mr. Davis, and described their actions and symptoms as stated in the early part of this report, but he also stated now that in the commencement of the disease many of them slobbered profusely; numerous small blisters appeared in the mouth and on the tongue, and that they then refused to eat hay or rough feed; that they manifested an inclination to lie in the snow, and on warm or sunshiny days they sought cool and sheltered places. On the 1st of January about 30 of them were lame and new cases developed daily for a number of weeks thereafter, also that some were yet taking the disease while others had recovered from it.

After the lapse of the first few days of sickness they regained their appetite and ate as well as ever.

The best animal in the herd of yearlings, which was also one of the first ones taken sick, died on the 5th of January; he refused to eat corn, frothed at the mouth, and suffered intense pain. The second death occurred on the 28th day of February; this was the calf, which died two days after the mother of it was taken sick. One died on the 8th of March; this one had been suffering for ten weeks, and had lost both hind feet at the fetlock joints. Three were killed. The 8 head which he bought since the disease broke out, all became sick within two or three days after they arrived on the place. About a week after I obtained the above history Mr. Keith's hired man told me that only 4 or 5 of the 8 head bought subsequent to the outbreak of the disease became affected. I also learned from Mr. Keith that in the early part of March he received the report published by your Department for the year 1880 and 1881, which contains a brief history and description of the foot-and-mouth disease; he read it carefully, and then made his first examinations of the mouths and states he found the conditions to accord exactly with the descriptions there given.

Mr. Keith had another lot of cattle, numbering 40 head. These were two-year-old steers and heifers, and a few cows. They were kept in a timber lot, separated from the yearlings by an ordinary rail fence, and run into a stalk-field and received the same kind of wild hay as the yearlings. These cattle were bought on or about the 1st day of November. On the 28th of February the first case of sickness appeared in this herd, 15 or 20 of which manifested symptoms of the disease (March 10). Nearly all of these cattle were in excellent growing condition.

On the 13th I separated all of the well cattle from those showing any evidence of the disease on the Keith farm, and had the sound ones corraled by themselves. I recorded the temperature of a number of the yearlings which were diseased, which registered as follows: 103.8°, 103°, 103.2°, 104°, 104.8°, 105°, 104.8°. A number of the milder cases registered 100.3°, 100.2°, 101.8°, 100°, 101.5°, 101°, 100.4°, 102°, 101.5°, 100.2°, 102°, 101.6°, 101.5°, 101.2°, 101°, 101.2°, 100.8°, 100.2°, 101.2°, 102°, 102.2°. (Here I broke my thermometer). This was quite a warm day, and I noticed an increase of temperature of nearly 1° over the tests of the 9th and 10th on the same animals. I found, out of 118 animals then on the place, 74 affected; of these 2 will lose all four of their feet; 4 have lost both hind feet; 9 have each lost one hind foot; 1 four-year-old cow has lost both hind feet and one front toe: 2 lost each one toe; 3 are affected in one foot; 6 in two feet, and 1 in three feet, all of which will probably lose the parts affected. The rest were lame in various degrees.

During my two weeks' observations among these cattle I found only the one case (the yearling red steer) which I could consider in any manner a recent case.

On this day (the 13th) I also examined the pond of water from which the cattle were in the habit of drinking, but found nothing contained in it to which I could attribute the origin of the disease, and from the history which I was enabled to obtain I could not discover any origin by contamination with foreign cattle. Yet it appeared to me that this outbreak was very evidently not so contagious as the foot-and-mouth disease is known to be, and I began to have very grave doubts about its being contagious at all, or its being the genuine epizootic aphthæ.

On the 11th, in company with Mr. J. W. Beard, Judge Thatcher, and Hon. Eli K. Titus, I drove out to the farm of Mr. Beard, and made a hurried examination of the cattle on his place, situated 2¼ miles north of Neosho Falls, in Woodson County.

I there found 5 animals, out of a herd of 75, affected similarly to those of Keith's and Goodrich's, although only one will lose both hind feet, and a second one one foot. Mr. Beard attributed the origin of the disease among his cattle to the introduction of the cow from Keith's, as before mentioned. As soon as he noticed any evidence of lameness he separated such a one from the rest of his cattle, and thereto attributes the small number affected among his herd. Two recovered from lameness after a few days. His cattle were fed on corn in the fodder and wild hay. The cattle drank from the Neosho River, which supplies good, clear water, and the approach to the drinking place is a gravelly bottom, free from mud. These cattle possess a timber shelter, with shed and hay-racks, and the feed-lot is sloping and well drained.

On the 18th I again visited Mr. Beard's place, accompanied by Prof. M. Stalker, of the Iowa State University. No new cases had appeared since my previous visit. Mr. Beard was absent, but we saw Mr. A. W. Orrill, the hired man, who made the following positive statement: That he came to Beard's place to work on the 16th of February; that on the following morning he noticed the old cow to be decidedly lame (this was the first one affected), and that on the 18th he assisted in bringing the cow from Mr. Keith's for which Mr. Beard had traded.

The Keith cow was the second one to show lameness, which was on the 22d. On the 1st of March, the third one; a day or two later, the fourth one was slobbering profusely at 4 o'clock p. m., and died the next day at 10 a. m. The fifth one became lame on the 5th, and several others manifested slight lameness, but have recovered therefrom. At this visit to Mr. Beard's we examined the feed and hay very closely, endeavoring to find some cause for the appearance of this disease. We found mixed with the hay a large proportion of the wild rye, and found this wild rye to to be extensively ergotized. This discovery at once accounted for the gangrenous phase of the disease. We followed up this clue with avidity, and went directly to Keith's and to Goodrich's place, and found the same conditions present among the hay there, but not near so extensively as at Beard's. I had examined the hay at Goodrich's several times, but was unfortunate each time in seeing hay in the racks which contained but very small amounts of the ergotized grass.

On the 14th I separated the Goodrich cattle, placing the well cattle into a field by themselves. I found 18 which had lost both hind feet; 5 had each lost one hind foot; 1 had lost both hind and one fore foot, and 1 was about to lose all four feet; 40 others were lame in one or more feet. This left 31 well out of the 96 head. No new cases had appeared in this herd during the time of my stay.

On this day I left for El Dorado, Butler County, at the request of Governor Glick, to investigate a disease among cattle said to be existing at or near that place.

After my arrival I called upon Dr. A. Bassett, mayor of the city. He told me that Mr. W. B. Collinswor h, residing 14 miles west, reported to him that he had a cow which appeared very stiff and lame, and that she had sores on the tongue. I drove out early in the morning of the 15th and examined the cow belonging to Mr. Collinsworth, and found her to be recovering from a mild attack of puerperal apoplexy. I returned to Neosho Falls on the same day. On the 17th I went to Hall's Summit, Coffey County, at the request of the county commissioner of that district. I was directed to visit the residence of Mr. George R. Smith, who had a cow which had lost some of her feet. I found the cow presenting the following conditions: The left forefoot had come off at the joint within the hoof, the left hind leg had broken off half way between the fetlock and hock joint carrying the lower end of the metatarsal bone with it, and the right hind leg was coming off at the same place. The right horn had also broken off close to the head. The cow was reduced to a mere skeleton, and was suckling a calf. Mr. Smith gave the following history:

On New Year's night the cow became cast by being tangled up in the rope with which she was tied; she was found by him in the morning; was loosened, when she got up and walked away. She ate and drank as usual. About a month afterwards she began to show lameness in her hind limbs; frothed a little at the mouth, and did not eat well. She gave birth to a calf on the last day of February. This cow was bought just before the holidays, and at about the same time he bought another cow. This second cow had a calf one day later than the diseased one. Both cows and calves have been kept in the same yard and on the same kind of feed, but only the one developed the disease. They were fed on chop feed and wild hay.

I returned again to Neosho Falls on the 18th. On the 19th, in company with Professor Stalker, I visited the Keith and Goodrich herds. On the 20th, in company with Dr. D. E. Salmon, of your Department, Dr. Stalker, Dr. G. C. Faville, of Colorado, and Dr. E. T. Haggard, of Lexington, Ky., I visited again the three affected herds, and also a fourth one owned by Christian Pribbernow, on Owl Creek, 12 miles south of Neosho Falls, Woodson County. Mr. Pribbernow owns 183 head of cattle, 54 of which are yearlings, 24 two-year-old steers, 15 three-year-old steers, 13 heifers with calf, the remainder cows and calves. At this place we found 16 affected, the symptoms and lesions being similar to those of the Keith and the Goodrich cattle. Seven of them will lose one or more feet, and the other 9 manifest lameness. Two or 3 which were slightly lame have recovered.

Mr. Pribbernow stated that the disease made its first appearance on the 15th of February, when several of the older cattle were noticed to be lame; soon thereafter swelling of the hind feet and extreme lameness appeared, rendering them unwilling to walk. He then placed 7 of the worst cases into a small yard by themselves. This yard was divided from the yard which held the yearling cattle by an ordinary rail fence. None of the yearlings became affected. The large cattle had been fed on corn-fodder and wild hay, and the yearlings in addition received millet hay and oats. The wild hay on this place contained a great amount of wild rye which was extensively ergotized.

On the 20th we saw the cattle at Beard's, Keith's, and Goodrich's, and found the conditions as herein described. After making these inspections, it was decided by Drs. Salmon, Stalker, Faville, and myself that we believed the origin of the disease among these four different herds of cattle to be due to the consumption of the ergotized grasses contained in the hay. But in order to more fully satisfy myself, I requested Dr. Salmon to accompany me to a farm lying adjoining that of Mr. Keith; therefore, on the next day, we drove out to the farm of Mrs. Dipple, which is situated west of Keith's. We there examined the hay and found it to contain a very small amount of the ergot. We also examined several of the cattle and found slight discolorations of the mouth, yet the cattle had never shown any lameness or indisposition whatever. We then looked over the well cattle on the Goodrich farm, which had been separated from the sick by me on the 14th, but found no new cases.

During the course of my investigations I killed 3 animals for the purpose of examination into the condition of the internal organs. In one I found enlargement of the heart and softening of the muscular walls; in another I found an infiltration of a purplish-colored fluid into the mesenteric glands, and in the third one I found no abnormal condition of any organ to be present. In all of these 3 I examined the alimentary canal very closely, but failed to find any pathological changes in its membranes or glands; these animals had each lost both hind feet. In carefully considering the conditions presented by all of these diseased cattle, and well weighing the value of the histories as given by the owners, I firmly believe that the disease may be called the "chronic" or "gangrenous form of ergotism."

Ziemssen says:

> It is not certainly known why in the one case the ergotism assumes the spasmodic, in the other the gangrenous form. But it seems remarkable that the spasmodic form formerly prevailed chiefly in Germany, while the gangrenous form was found principally in France, and there particularly in the Sologne. But this difference does not hold universally, for epidemics of the gangrenous form have appeared also in Germany, Austria, Russia, and Sweden. It is not known whether this variety depends upon the difference in the activity of the ergot in different seasons, or whether such changes are the result of some peculiar property of the ground in which the corn grows. In the Sologne it was generally the ergot of maize which produced the poisonous symptoms, whereas in Germany the ergot of rye was almost exclusively mentioned as the cause of the disease. It is, however, very improbable that the difference between these illnesses is dependent on the different parent plants, because at least

therapeutically the same effects can be produced by the ergot of maize as by that of rye, when the quantities are equal. It is most probable, then, that there is a simple quantitative difference in the absolute and relative quantity of the poison taken into the system.

Of the action upon the human system the same writer says:

> The poison of this drug (ergot) has a special and most powerful action upon the skin. Besides very abundant perspiration, pustules often break out, or even larger furunculi. The exanthemata sometimes resemble scabious eczema. They appear in the later stage of the malady; as, *e. g.*, in Aschoff's case, fourteen days after the first appearance of symptoms of poisoning. But other disturbances of nutrition in the peripheral organs are also reported, as, *e. g.*, whitlows on the fingers, occurring as late as the fourth and fifth week, and diseases of the finger nails, which are encircled by a dark ring. Cardiac contractions are generally slow and feeble, the arteries are constricted and contain little blood. The respiration is very labored during the spasms, but tolerably regular in the free intervals. When death supervenes it is usually not until after a fortnight or later; the convulsions may have ceased, yet loss of sight and hearing, with violent headache, stupor, and delirum, may set in, attended with diarrhea; and thus the fatal stage may assume the form of typhus and general collapse. Death is generally ushered in by either convulsions or paralytic symptoms. The whole form of the illness, therefore, is very variable, and its course highly irregular. The illness may last four to eight weeks, and even longer.
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> The symptoms which characterize gangrenous ergotism as such, often appear within from two to seven days, but are frequently delayed for two and three weeks. An erysipelatous redness shows itself on some spots in the periphery, most frequently on the toes and feet, but also on the fingers and hands, more rarely on the ears and nose; soon after, the epidermis is raised like a bladder by serous exudation; the ichorous contents of this are soon discharged, and a gangrenous spot more or less large is left. Then dry gangrene develops very rapidly at the affected spot.
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> The part affected is very painful while the redness is invading it; but later on it becomes quite insensible. The gangrenous spot may exhibit either the dry or moist form, according to whether the discharge was checked or encouraged; upon this also depends the greater or less intenseness of the odor or putrefaction. In some cases the gangrene was limited to one or more toes, sometimes only to single phalanges; in other cases, however, the entire foot or hand was affected; not infrequently the gangrene extended to the trunk; it was possible for the patient to lose both feet or both arms. Indeed, a few cases are reported in which all four extremities were lost. The gangrenous parts become separated from the healthy tissue by a well-defined line of demarkation, and the affected part may fall off itself or must be removed by an operation. This process of demarkation is often attended with serious disturbances of the general condition of the patient; sometimes a modified form of continued fever is developed followed by phthisical changes; in a few cases, from absorption of ichorous matter, pyæmia and septicæmia set in, and are, of course, fatal. When the gangrene was confined to parts of minor importance, the patients usually recovered; greater losses were naturally more frequently fatal. In some cases obstinate diarrhea brought on marasmus and death, even when the extent of the gangrene was not very considerable. We must mention, however, that in many cases the diseased process did not advance beyond the erysipelatous redness; marked cyanosis may be observed, and yet a separation may take place and the circulation be restored. The duration of the entire illness varies, and may be protracted through several months. In favorable cases the course is ended in a few weeks.
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> This form of gangrene, like all other forms, depends on the fact that the part affected is deprived of its blood supply, and its nutrition thereby arrested; consequently, it must pass into a state of decomposition. The only question which can be advanced here is whether it is inflammation which leads to gangrene, or whether the process is of a non-inflammatory character, resembling that which occurs when all the vessels going to a limb are ligatured. When we consider that the initial, so-called erysipelatous redness is simply dependent on the cyanosis, and that these spots are not, as in a case of inflammation, hot and swollen, but, on the contrary, they become very cold, and warmth cannot be restored in them, and that the affected limb is not at all swollen, the hypothesis that such a gangrene is of an inflammatory character must, *à priori*, be rejected. When we further reflect that there is no fever at the outset, the second hypothesis becomes still more probable. Exclusion of an extremity from its ordinary blood supply is quite conceivable from our current views of the action of ergot on the vessels and the distribution of blood.

On the 10th of April I was requested to go 8 miles north of Sterling to the residence of Mr. John Kratz, who said that he had a number of cattle affected with lameness and sore feet. I proceeded to his place at once, and examined his herd of cattle, numbering 30 head. I found 11 cows and heifers more or less affected with excoriations in the clefts of their feet. In several cases I found these excoriations to extend around the bulb of the heels, causing a separation of the horny walls at the coronet from the fibrous structure of the foot. In one cow, twelve years old, I found considerable ulceration at the bulb of the heels of both fore feet; the matter burrowing beneath the horny wall caused a destruction of the wall to the extent of 1 inch downwards, and the imperfect formation of an inch of the horn at the front part of the feet indicated that she had been affected for two months or longer. These excoriations discharged a semi-liquid matter, which gave off an odor like to that of thrash in horses' feet. Mr. Kratz stated that two of the cows walked with difficulty for several weeks, and that for a week or more previous to their lameness he detected the peculiar odor arising from the feet while milking. On the 13th of March he bought a thoroughbred short-horn heifer, and one week after he brought her home she also became lame. When I saw her she was affected in all four of her feet, and was exceedingly lame. All of his cattle, with the exception of two that were over two years old, were affected. In none of the feet was there any swelling, but they were extremely sensitive to handling. The disease appeared as frequent in the fore as in the hind feet, and in several cases three or all four of the feet were affected alike. The majority of them became lame while the ground was yet frozen up. Mr. Kratz has one of the finest bank barns in the county, and takes excellent care of his stock. He stables his cattle at night and on stormy days; they have good bedding at night, and the stalls are cleaned daily. In pleasant weather the cattle were turned into the barn-yard. This yard is covered almost daily with clean straw to be worked into manure. The yard is sloping and well drained, and the cattle drink well water from a trough laid in the yard. During the winter the cattle received ground feed, timothy, and clover hay, but about the 1st of March he began to feed rye bran and second-crop meadow hay; the latter contained a large proportion of blue or June grass, and an examination of it revealed that it was ergotized; almost every seed capsule contained the fungus. If the ergot in the hay did not produce the disease I am unable to account for its origin. The cattle were not in a plethoric condition; the yard was free from mud, and they were well housed, and the stables kept scrupulously clean.

The disease was what would commonly be called foul in the foot. I treared them by cleaning out the clefts of their feet, applying carbolic acid solution and tar secured to place by a wad of oakum and bandage. In ten days recovery was complete.

Mr. Thomas Comboy, of Hume Township, had 2, and Mr. Edward Tyne had 1, affected similar to those of Kratz.

Respectfully submitted.

M. R. TRUMBOWER, V. S.

STERLING, ILL., *March* 27, 1884.

INTERNATIONAL VETERINARY CONGRESS.

REPORT OF DR. JAMES LAW.

SIR: I have the honor to submit the following report on the International Veterinary Congress at Brussels and the European Veterinary Schools:

In accordance with the call issued by the committee of arrangement, the Fourth International Veterinary Congress met at Brussels on September 10, 1883. There were present 311 veterinarians, of whom 218 were Belgians, and 93 from other countries of Europe and America. The congress remained in session seven days, and engaged in the discussion of the following subjects:

1. THE ORGANIZATION OF VETERINARY SERVICE.
2. CONTAGIOUS PLEURO-PNEUMONIA OF CATTLE.
3. EDUCATION IN VETERINARY MEDICINE.
4. THE RIGHT OF THE VETERINARIAN TO FURNISH MEDICINES.
5. PULMONARY PHTHISIS.

1. THE ORGANIZATION OF VETERINARY SERVICE.

This subject was ably presented by Zundel, of Strassburg, the reporter of the committee appointed to introduce it.

The veterinary profession can no longer be estimated only or mainly by its knowledge of therapeutics and its success in curing disease, otherwise it would be to the pecuniary interest of the veterinarian to keep the community in ignorance of the causes of diseases, epizootic, and sporadic, while he fattened on the proceeeds of an extended practice. But to-day it is the glory of the veterinary profession that it is pre-eminently a sanitary and preventive body. It can point to day to the comparative absence from European flocks and herds of those plagues which but a century ago desolated the countries at frequent intervals; it can show pastures now salubrious, which were formerly pregnant with the seeds of death; it can offer immunity to the system from poisons whose touch was heretofore deadly; and it can show how to extinguish in animal hosts the causes of disease, which, when conveyed to man, would entail extended suffering and death.

By his knowledge of zootechny, the veterinarian contributes to the improvement of the different races of domestic animals; by his knowledge of sanitary police he protects the purchaser of animals against imposition, and knowing how to exclude epizootics he protects international commerce, which the constantly increasing facilities for communication render daily more important; by his special knowledge of animal diseases he can insure that maladies intercommunicable between animals and man shall not be conveyed to the latter through meat and other animal products.

The question of the organization of the veterinary service as a sanitary branch of every government had been extensively discussed at the Third International Veterinary Congress at Zurich in 1867, and at the following national veterinary conventions: For Germany, at Frankfort-on-the-Main, in 1872; for France, at Paris, in 1878; for Italy, in Bolonge, in 1878; and for Belgium, at Brussels, in 1880.

The Zurich Congress pronounced that:

1. The practice of veterinary medicine should be regulated by law.
2. The veterinary art ought to be an integral branch, but independent, of the sanitary administration.
3. Veterinary medicine should be represented by competent persons closely related to the authorities; inferior, medium, and superior.
4. No one should be allowed to practice veterinary medicine unless he has pursued the required studies in a public veterinary college, and has acquired after examination the diploma or the legal character of veterinarian.
5. All veterinarians may be called as experts by private individuals, but the judicial authorities ought only to call as competent persons veterinarians who are officially recognized.
6. In cases of sanitary police the administrative authorities ought not to have recourse, save exceptionally, to those who have not been officially recognized as competent.
7. The expression "competent person" should be understood in the sense that he is a diplomaed veterinarian.
8. All veterinarians ought to have the right to furnish the medicines required in their practice, but always under a suitable control.

The four national conventions pronounced in similar terms for a guarantee of education and competency on the part of the veterinarian, and for the organization of a sanitary veterinary service in each state, which should deal with epizootics, advise how to render districts salubrious, and how to improve the races of domestic animals, and inspect abattoirs, slaughter-houses, and rendering works. The German convention pronounced in favor of a unification of the laws of the different German states in regard to veterinary sanitary police, and veterinary jurisprudence, and for the frequent publication of statistics of contagious diseases and mortality. The French convention pronounced in favor of including in the sanitary corps all the veterinarians practising in a department, and for the election of a departmental veterinary director by their votes.

In the spirit of these antecedent conventions the following resolutions were submitted:

1. To organize in every country a veterinary sanitary service charged exclusively with all that pertains to veterinary science, and the members of which shall be counsel of every department of the Government, but which shall be more especially represented directly in relation to the central power—that veterinary medicine shall there have her chief of service.

2. The veterinary sanitary service should utilize the greatest possible number of veterinarians. To do this efficiently and economically there should be recognized two degrees or classes of veterinarians. The one of a local nature connected less with the state than with the municipal and provincial authorities and charged with the surveillance of fairs and markets of animals, the inspection of meats of the butchery and abattoires, the control of rendering works, the inspection of breeding animals, the inspection or direction of mutual insurance companies against the mortality of animals, the revision of the census of domestic animals, &c.; the second charged with a wider range of duties, comprising the service of the state, and capable of becoming international, embracing especially the suppression and prevention of contagious maladies and epizootics, and also the control of the different other veterinary corps.

3. Between the various states which by a regular repression and preventive serv-

ice against epizootics, can furnish the guarantees of a good veterinary sanitary police there should be established a treaty having for its object: 1. To apprize the other states as speedily as possible of any eruption of Rinderpest, pleuro-pneumonia contagiosa, aphthous fever, sheep-pox, maladie du coit (dourine), glanders (or farcy), and of scab in sheep. 2. To publish periodically a sanitary bulletin upon these diseases, their extent, progress, and extinction, which documents should also be published in the international bulletin if judged necessary. 3. To oppose these diseases by measures of sanitary police which have first been discussed and adopted as the most advisable. 4. To furnish with animals and herds that are moved into and out of a territory certificates of origin and health of a guaranteed administrative value. 5. To contribute to the publication of an international veterinary sanitary bulletin.

FIRST RESOLUTION.

Among the arguments advanced in support of the first resolution were the following:

In various countries of Europe and America these duties essentially veterinary are left too much in the hands of persons ignorant of veterinary science. In some they are largely dealt with by bodies mainly medical—sanitary boards. No one denies the great debt of veterinary medicine to that of man. The works of the physician in anatomy, physiology, pathology, surgery, &c., furnish the most valuable material for the medicine of animals. The principles of both branches of medicine are the same. The observations and experiments on animals made to advance the one are of the highest value to the other. Many of the parasites and zymotic poisons of man are common to the animal as well. But when we come to the application of these principles to the diagnosis and treatment of disease in animals the physician finds that the two medicines diverge at every step, and that without a special training he is helpless to deal with that of animals. At one time physicians could be found who were more accomplished in microscopy, &c., than the veterinarian, but to-day in the veterinary schools of Europe the subjects receive as ample attention as in the medical.

In some cases the bureaucratic element tends to the subversion of the veterinary, and men whose only knowledge of veterinary matters come from consulting the writings of some one veterinarian will pass judgment upon the advice of the most skilled expert, and even decline to employ it. It is for the jurist to formulate and promulgate the laws, but it is his duty to base these on the best counsels of veterinary medicine.

To maintain a satisfactory system not only must the veterinary sanitary work in départments and cities be carried on by veterinarians, but there must be at the seat of power a veterinarian or commissioner of veterinarians as a centralizing point for all that relates to the veterinary service, and for the direction of that service. As countries in which such centralization exclusively veterinary has been secured may be named Holland, Denmark, Sweden, Russia, some of the southern German states, Saxony, Bavaria, Baden, and Alsace-Lorrain, Portugal, and Belgium. In Austria there is a consulting committee on epizootics, but which occupies itself with prevailing maladies alone. In the dependent Austrian states the Landesthierarzt is a simple member, sometimes a simple agent of the sanitary council. In Prussia there is not even a chief veterinarian in relation to the minister, who only consults the technical deputation in which veterinary science is represented. In each provincial government of Prussia a departmental veterinarian or assessor is attached usually to the medical council as councilor (referee) for veterinary service. In France the veterinary element now predominates in the superior committee of epizootics. In England the privy council veterinary department has three veterinarians in a body of six members. In Switzerland, attached to the department of agriculture, is a veterinary commissioner of epizootics as counsel on the whole subject of federal veterinary police, but he has no voice as to the competence of cantonal authorities. In Italy there is no central veterinary officer, and the bulletin of epizootic statistics is compiled from data furnished by municipalities.

After considerable discussion the following was passed with only four dissenting votes:

> To organize in each country a veterinary sanitary service, charged exclusively with all that pertains to this service, of which the members, all veterinarians, shall be councilors of all branches of the administration, but which shall be especially represented near the central authorities, where the chief veterinary official shall be stationed.

SECOND RESOLUTION.

In regard to this the reporter pointed out the impropriety of binding the Government to employ as its local expert the veterinarian who happens to be in attendance upon the infected herd, the danger of such a person carrying infection to other herds in the round of his practice, the suspicion, usually unmerited, that he may hide infection in the interests of his employers rather than apply rigid measures of extinction for the good of the commonwealth, the fact that he may be an excellent general practitioner and yet not a specialist in epizootics, who may counsel treatment when the best sanitary science imperatively demands sacrifice, who will make a dangerous distinction between farcy and glanders, or who will prefer preventive inoculation in pleuro-pneumonia to slaughter. What can be expected of the veterinarian who has become hopeless and apathetic, whose calling is to him but a handicraft, or who has become morally debased?

The increasing numbers of live stock, the facility in transporting them long distances by steam, and the great demands of Western Europe for outside supplies demand for the inspector of to-day a very different official from those of the past. The lack of a thoroughly efficient veterinary sanitary service was felt when the lung plague attained such a wide extension in 1840, when the Rinderpest ravaged Holland and England in 1865, and France in 1870, when influenza spread over America in 1872, lung plague in 1878, or when hog cholera destroyed in a single state hogs to the value of $20,000,000 in one year.

Already a good beginning has been made in different states. Holland has nine district veterinarians under state salary. France has in the department of the Seine five exclusively occupied in the state service; England has twenty-three salaried port inspectors; Portugal has twenty-one on state salaries, not large enough, however, to make them independent of practice, and the same applies to the states of Germany and Austria where the Government veterinary officers have regular salaries, but insufficient for their entire maintenance.

The field with which a departmental veterinarian is charged should be large enough to insure that the office shall be no sinecure, and that he shall not by private practice interfere with that of the local veterinarians. He should be appointed after a special examination, or exceptionally for signal services in sanitary police.

The department veterinarian should counsel the central authorities, advise legislation, take the direction in the prevention and extinction of epizootics, and direct the work of the local veterinarians. To these last would be left the inspection of fairs and markets, meat markets, slaughter-houses, &c., of animals sent by railroad, and the general local work of the service. They should make to the chief veterinarian written reports of any extraordinary occurrence in their district and periodic reports of the general work accomplished. These will furnish data for the publication of statistics of the animals kept, bred, purchased, sold, killed for food, dying of sporadic and epizootic disease, attacked by epizootics, &c. These in relation to geology, soil, drainage, meteorology, culture, breeds, breeding, alimentation, use, &c., will supply data of the greatest value to stock holders, veterinarians, hygienists, physicians, dealers, and political economists.

As a rule the destruction of animals to arrest an epizootic should be ordered by the chief or departmental veterinarian, thereby securing at once the guarantee of a diagnosis by more than one expert and saving the local veterinarian from the unpleasant duty of ordering a work that may be unacceptable to his regular employer.

Not the least important duty of the local veterinarian is in connection with the mutual assurance of animals against deaths from sporadic diseases. Losses from epizootics are indemnified by the state, but losses from diseases that are not communicable justly become a tax on the stock owners of the municipality or district. Such an arrangement engages the interest of the stock owners not only to report the contagious diseases early, but the non-contagious as well, and to use all resources of science for the cure of the latter, but much more for their prevention. The local veterinarian as a regular officer of such societies, directing, supervising, inspecting, finds his interest consulted in urging all that can conduce to health in breeding, management, and treatment, and wherever such associations have been introduced there has been a notable improvement in all that pertains to the quality and numbers of the live stock.

The local veterinarian, being an essential part of the national veterinary sanitary service, it should not be in the power of the local authority to discontinue his services, nor to appoint to the position any one but a competent veterinarian (a graduate).

After discussion mainly of the right of the sanitary veterinarian to engage in ordinary practice, and of the desirability of specifying two classes of veterinary officials, the following was adopted by a large majority:

> 2. That the veterinary sanitary service should utilize the greatest possible number of veterinarians. It embraces the inspection of fairs and markets of animals, of meats of the butchery and abattoir, the control of rendering works, the inspection of breeding animals, the supervision or direction of mutual assurance societies against the mortality of beasts, the revision of the census of domestic animals; * * * it comprehends the service of the state and may become international, embracing especially the repression and prevention of contagious maladies and epizootics, also the contro of all other veterinary service.

THIRD RESOLUTION.

In support of the third resolution Zundel advanced that contagious maladies are no longer recognized as of spontaneous origin; that day by day the advocates of spontaniety yield the point, and that well-observed facts prove that it is by contagion that these maladies are at once propagated and perpetuated. To prevent the propagation of these maladies it is necessary to have in all countries enjoying an international commerce in live stock a common, permanent, and efficient system of preventing or at least of arresting them in their inception. The value of an international arrangement for the control of Rinderpest has long been recognized and becomes yearly more important. The demands of Western Europe for a meat supply are constantly increasing, while the animal production in Western Europe remains stationary, or even diminishes. This imperious demand for beef, and the facility for its supply by cheap railway transport from the East, cannot fail to insure an increase of the Eastern traffic, and unless conducted under efficient international measures of protection this must deluge the West continually with this most fatal of all bovine plagues. The improved culture on the Steppes and the introduction of better forage plants enable the stock owners to tide over the dry summer and the frozen winter more satisfactorily, and thus contribute still more to the numbers and excellence of the Eastern supply. Austria imported 20,000 Russian cattle in 1861, 30,000 in 1868, and 55,000 in 1872. The more stringent restrictions have later lessened the numbers, but the increasing demands of the West and supplies of the East must, ere long, turn the tide once more, and bring large installments of these Eastern beeves. Hitherto protection has been sought by the more or less perfect exclusion of Steppe cattle, but the time must come when this shall be superseded by an international arrangement founded on solid guarantees of the soundness of the cattle exported.

Already in regard to Rinderpest this has been attempted; to-day all the different countries of Germany act on the same law, that of 7th April, 1869, in repelling and repressing this plague, and in April, 1872, Austria called in Vienna a conference to consult as to the requisite international guarantees, and delegates attended from Germany, England, Austro-Hungary, Belgium, France, Italy, Roumania, Russia, Servia, Switzerland, and Turkey. If each country would organize an efficient service to stamp out Rinderpest as far as possible and to prevent its radiating outward from any existing center of infection, every state might by this perfect isolation of its limited infected area secure an untrameled cattle traffic for its entire territory besides.

The same can be done for contagious pleuro-pneumonia, and it is easily demonstrated how much evil has already resulted from the neglect of other Governments to respond to the Swiss movement in this direction in 1876. From this England loses yearly about 5,000 cattle; Belgium, 2,000 to 3,000; Prussia, 2,000; Wurtemberg, 500; Austria, 2,000 to 3,000, and France and Italy corresponding numbers. Rinderpest is comparatively easily suppressed, because its prompt eruption and fatal issue strikes the population with terror and it cannot be hidden; but the lung plague strikes slyly, hides its tracks, and, creeping into the stables unseen, it diffuses its poison, infects, benumbs, and paralyzes the lungs without the body appearing to suffer, and it only manifests itself by outward symptoms when all is lost. More than this, the lung plague often assumes the benign and almost latent form, so that after months of incubation it still rests unrecognized and unsuspected, assuming a chronic type, but still scattering the poison, and the subject even appearing to recover, without an abatement of its infecting power. Often, too, the laws, and even very recent ones, take but half measures against this plague, leaving it to intrench itself more and more firmly and to maintain itself permanently in a country where it is only an exotic, a foreign invader, and where it could easily be excluded through an efficient international system. For this, therefore, even more than for rinderpest, an efficient system of mutual international protection is urgently demanded.

The aphthous fever is largely combated by restrictions on the movement of cattle and the interdiction of fairs and markets, yet these rarely arrest its progress, but the barriers that put a stop to the rinderpest allow the aphthous fever to pass, as happened in Saxony in 1867. With such a diffusible poison success must be sought in its declaration, suppression, and seclusion in the home herds and by preventing it from passing at all into the channels of traffic, and this can only be secured through a common international system.

Formerly lung plague, aphthous fever, and sheep-pox remained habitually circumscribed in particular districts; to-day, with the great movements of stock in mass, their concentration in vast markets, and their constant changes in the large feeding stables, it has become impossible to deal effectively with these plagues except in native herds, and this imperatively demands a uniform international system, with solid mutual guarantees.

Glanders and farcy occurring in an occult form with lesions internal and unsuspected, and following a chronic course, is another fruitful source of trouble, and a country that pays for such animals when slaughtered especially suffers, as the diseased animals are smuggled across the frontier in order to secure the indemnity. Even the residence of several months required in order to the payment of indemnity is insufficient to guard against this sharp practice.

The mad dog does not always take the road to the custom-house, nor recognize the colors of the frontier posts. Here, therefore, it becomes necessary to maintain a common system of repression and the mutual notification of the existence of rabies.

Maladie du coit, though unlike the two last, in that it respects the human family, should yet as regards the equine races be made the object of international guarantees.

Anthrax and several parasitic maladies, including even measles and trichinosis in swine, are more purely matters of local danger, and are to be controlled by local measures and by inspection in the great meat markets.

Energetic sanitary measures within the limits of a single state have been often notably successful, as against lung plague in Switzerland; Holland, Portugal, Sweden, and Denmark against sheep-pox, apart from Northern Germany and Hungary, and against rabies in Baden. How much better if there were a common international co-operation, which would at once more effectively repress animal plagues and release the commerce in live stock and all their fresh products (hides, hoofs, hair, bristles, fat, guts), as well as fodder and straw, from the present obnoxious and injurious restrictions.

Unfortunately in all modern legislation on the subject not only are the laws peculiar to the individual state, but they reflect the jealousies and lack of adjacent states. Thus in the absence of official trustworthy and trusted reports a whole country has its trade restricted, delayed, and seriously injured for a slight outbreak which could easily be sequestrated and a perfect guarantee of its non-extension furnished. It is to be feared, indeed, that in some instances what is practiced under the name of veterinary sanitary police is conceived more in the spirit of maintaining the high price of meat than of restricting and extirpating animal plagues.

If the work could be made international, and if the plagues could be effectively dealt with in the local areas of their prevalence, all the more obnoxious interference with commerce might be done away with, the present temptation to clandestine trade in infected animals obviated, and a better and more reliable protection afforded.

The inspection of animals at the frontiers has proved practically useless, because animals that have become infected but do not yet show signs of disease are necessarily allowed to pass, and with the modern immense railway traffic in fat animals a sufficient quarantine is practically prohibitory of importation.

Another grave objection to this system is that animals found diseased are simply sent back over the frontier, and as the service is national and not international, the neighboring state is not warned of the active focus of infection thus created within it.

Certificates of origin and health emanating from officials and based on expert knowledge of the sanitary condition of the district furnishing the stock should be really valuable documents, whereas certificates made out by irresponsible individuals, and with which the smugglers are now most numerously supplied, are grounds for suspicion rather than confidence.

The following sum up the principles which ought to dominate in an international veterinary sanitary service:

a. Each state ought to notify the governments of adjacent states of those joining the international agreement, and of all that desire it, of every outbreak of rinderpest, lung plague, sheep-pox, *maladie du coit*, glanders, and rabies, its exact locality and extension; and this should be done as quickly as possible, even by telegraph.

Other grave maladies transmissible and importable, and especially the typhoid affections and sheep scab, should be made the object of extraordinary precautions and mutual notification.

The authorities ought to carefully investigate the channel by which contagion finds its entrance and is propagated; and the officials of the country from which it was derived should be furnished with the information necessary to enable them to trace it to its earlier sources.

Each Government ought to publish in its official organs a sanitary bulletin upon the sanitary states, the progress of epizootics, the measures enforced, the interdictions of imports, the changes made in these prohibitions, and of the suppression of the plague when that has been effected. This bulletin should be sent to editors of official journals of states that request it or that have signed the agreement.

b. The authorities of frontier districts ought to notify directly the authorities of neighboring districts whenever rinderpest or aphthous fever has been detected within 100 kilometers (62 miles) of the frontier, or when lung plague, sheep-pox, glanders, or rabies has appeared within 50 kilometers (31 miles) of the frontier. In rinderpest, sheep-pox, and rabies the notification should be made by telegraph.

c. Every state should so organize its veterinary service as to be able to rapidly stamp out rinderpest or other contagious maladies.

d. Every state should provide that the laws of sanitary police should be rigorously enforced.

e. By previous arrangement the laws of sanitary police in different states should be almost identical in measures of isolation, veterinary surveillance, sequestration, removal and burial of carcasses, and in disinfection of all persons and animals, of objects, clothing, and harness, stables, and of railway cars that have carried animals or animal products.

f. For all animals attacked or suspected of one of the contagious maladies above mentioned, and killed by police order. an indemnity should be paid, which each government may fix by special law, but which should in no case be less than half the value of the animal if it had been sound.

g. A previous arrangement should prescribe the principles which ought to control the international relations, the roads and means of transport to be taken, the conditions of movement of stock, and, above all, that which refers to certificates of health and origin, which ought as much as possible to be uniform in the different countries, and for the important assignments viséd by the consuls of the respective countries.

h. The laws of sanitary police should order the transporting agent to make instant declaration of diseases that may supervene in transit, and should prescribe the measures of sanitary police applicable to them.

i. There should be published an international sanitary bulletin at intervals of fifteen days, and giving the sanitary condition of each country according to the special bulletin, which by international agreement each government should publish. The international guarantee should be based on government morality, on a spirit of justice, on practical reason; thus commerce will acquire that freedom and integrity which are essential to it. It should prescribe the movement of all stock coming from a country, the sanitary condition of which is unknown. The great publicity given to the official reports, often sent by telegraph and diffused through the most rapid channels, would give a guarantee of their veracity; they ought to emanate from the authorities of the countries and be drawn up by veterinarians.

With this bulletin they would know at each custom-house what countries can supply stock to international markets, and from what the stock must be refused; this international bulletin should be a check on certificates of health and of origin, and should enable the officials to appreciate their value.

The bulletin would thus furnish the best mutual guarantee for international commerce, and give mutual confidence between nations carrying on with each other a traffic in domestic animals.

The nations will better comprehend on the basis of epizootics what they generally fail to arrive at on that of politics. One step may perhaps lead to others.

The third resolution, adopted unanimously, reads as follows:

> Between the different states, which by a regular service repressive and preventive of epizootics are able to furnish guarantees of a good veterinary sanitary police, there should be established an agreement having for its object: 1. To notify other states, with the least possible delay, of the eruption of lung plague, aphthous fever, sheep-pox, *maladie du coit*, glanders (or farcy), and sheep-scab. 2. To publish a periodic veterinary bulletin on these maladies—on their status, extension, and terminations; which information should also be sent to the international bulletin, if judged necessary. 3. To combat these maladies by measures of sanitary police which have been previously discussed and adopted as the best. 4. Not to permit the delivery of animals nor of herds which are being sent in or out of the territory, except on certificate of origin and of health of a guaranteed administrative value. 5. To contribute to the publication of an international veterinary bulletin.

REMARKS.

The above has been reported at considerable length because of its manifest bearing on the problems which now press on the American people for solution. In America, as in Europe, the medical and political authorities have undertaken too much of what properly pertains to the veterinary profession, and, as a consequence, we have had measures that aimed at repression rather than extinction of animal plagues, and the administration of these and even of better measures has been made in such an irregular and badly sustained manner that what had been gained to-day by much effort and outlay was lost on the morrow by a relaxation or entire suspension of the rules.

To have efficient legislation the best available veterinary advice should be secured as to the measures to be framed in a bill, and a veterinary authority should be intrusted to see the statute rigidly and impartially administered. Place it in the hands of a mere beaurocracy and this will too often, as in the past, result in its administration in what they consider the spirit—not the letter—of the law, until all its valuable provisions are frittered away and lost. A knowledge of the diseases is essential to a knowledge of the fundamental principles on which suppressive measures must be based, and, in ignorance of these principles, any attempt to carry out the law in its spirit rather than its letter is sure to end in blundering and failure.

Then, again, as regards interstate administration, nothing can be more instructive to Americans than the European difficulties in dealing with the animal plagues under the modern conditions of great manufacturing prosperity and the unprecedented activity of the traffic by rail. Examinations of cattle in transit must be given up as absolutely inefficient, for herds that formerly developed disease during the tardy and toilsome movement by highways can now be sent through in a few days by rail and long before the deadly germs within them can manifest their presence by overt symptoms. No better commentary can be furnished on the utterly futile provisions in a number of successive Congressional bills for the inspection of cattle in transit in our inland trade and of beeves about to be shipped to Europe. Again, the enormous proportions of the constantly increasing cattle trade toward mining, manufacturing, and commercial centers in Western Europe has been found to be utterly incompatible with the maintenance of such a quarantine as would protect against imported contagion, and they are for the first time brought face to face with the fact that the protection of any country in the line of this great cattle traffic must be secured, if at all, by a perfect system of seclusion and suppression in the country which furnishes the supplies of live stock, and by an honorable exclusion by such country from the channels of traffic of all live stock and their fresh products having their origin in an infected district. American lawmakers and administrators must see that our uninfected States and Territories can have no guarantee of continued safety in our present system under which live stock from infected States and districts, without even the pitiful and comparatively useless form of inspection and certificate, are sent to the very source of our great live-stock traffic. Fortunately for us our westward traffic in live stock is so limited that it is quite possible to impose an effective quarantine on all subjects moved in that direction. If nothing more can be done, this at least is within our power. Each State can quarantine all cattle or other live stock from an infected State, or if the State fails in its duty to itself and neighbors the United States can step in and regulate this item of intestate commerce.

Again, the experience of Europe with her great live-stock traffic by rail shows how utterly helpless we would be in any attempt to control these animal plagues if they once reached the source of this traffic. As is now the case with Texas fever, our first resort would be to prohibit all movement of susceptible stock from the infected areas, and the next to seek by every possible means to stamp out the infection on the native herds. If Europe with her extensive, not to say universal, fields of infection, finds her profit in maintaining a uniform veterinary sanitary service, operating at every point over the broad continent, and suppressing and secluding animal plagues wherever they may appear, how much more profitable must it be for the United States in dealing with her one exotic and imported plague, that of the lungs of cattle, which is still confined to the merest strip of her territory, and when the proposed control is to prevent its extension over the whole continent, and the necessity for a similar service and control from the Atlantic to the Pacific? It would require but the outlay of a sum equal to half our yearly losses on cattle exports by this cause alone to abolish this cause forever; it would require but the expenditure of a trifling fraction to save us from the future loss of millions. Again, if the independent nations of Europe find it necessary to have an international system of repression and extinction to expel their prevailing animal plagues, and if they have to sink national jealousies and rivalries in the presence of these international enemies, will the United States of America, with a Federal Congress and one Federal Executive, sacrifice to a sentiment our birthright to the most extended live-stock interests in the world?

Shall we calmly see the European states, with a legacy of ages of warfare and mutual hate, and ground down by their immense standing armies, the root and fruition of their common suspicions, unite cordially and loyally in a common international work to crush out the prevailing infections of centuries and to secure an untrammeled traffic in healthy live stock and sound meat while our mutually dependent States, bound in one federation, an unit in war, an unit in commerce, and an unit in all that relates to foreign nations—shall these States let a mutual jealousy prevent an interstate sanitary work court the general diffusion of our exotic infections, load the continent with animal plagues under which modern conditions must be more ruinous even than those of Europe in the past, and shut themselves out from supplying the meat market of the world which it is now theirs to take and to hold?

2. CONTAGIOUS PLEURO-PNEUMONIA OF CATTLE.

This subject was introduced by an extended report by Professor Degive and two supplementary reports by Messrs. Leblanc and Putz. Degive arrived at the following conclusions:

A.—DIFFERENTIAL DIAGNOSIS.

1. From an anatomical point of view we may consider as contagious and epizootic all interstitial pneumonias of a certain extent of which the development does not depend on local conditions or causes.
2. From a physiological stand-point, epizootic pleuro-pneumonia is especially characterized in the living animal by its contagious character and the symptons of lobar pneumonia.
3. In an infected stable every animal that shows fever with one or more symptoms denoting irritation of the respiratory organs, cough, hurried, plaintive breathing, &c., should be suspected of pleuro-pneumonia.
4. The spontaneity of one case of pleuro-pneumonia will not exclude the existence of the contagious affection.

B.—PROPHYLAXIS.

1. The development of pleuro-pneumonia may sometimes be prevented by a simple recourse to go d hyg eni conditions.
2. Animals affected with the malady, or suspected of it, should be sacrificed as quickly as possible.
3. Animals suspected of infection, or very much exposed to contagion, should be isolated or sacrificed. The slaughter of animals suspected of infection is more especially indicated when the disease manifests itself very exceptionally, or for the first time, in a stable belonging to a commune rich in cattle.
4. Animals suspected of infection, or very much exposed to contagion, and not sacrificed, would be profitably submitted to inoculation or to an appropriate preventive medication. Inoculation is especially applicable when the animals make part of a large herd, subject to frequent changes, or where the malady has already appeared several times.

5. Inoculation should not be prescribed as a general or obligatory measure until a method is devised which can be practiced without danger to the health or life of the beasts operated on.

6. Inoculation should not be practiced without the previous authorization of the local authority. It should only be done by a veterinarian and under police surveillance.

7. Every inoculated animal should be considered as suspected of contamination and treated as such.

8. The diseased and suspected animals should be reported to the authority with the least possible delay. This report should be made obligatory on owners and attendants, veterinarians, and inspectors—the experts of the abattoir or butchery.

9. Quarantined (isolés) animals should be made the object of a special census and should receive a distinctive brand with a hot iron.

10. No animal suspected of infection should be moved without previous authorization of the communal administration. The permit to move should only be granted for animals destined to the butchery; it should only take place in special conditions, under the supervision of the police and in such a manner as to prevent all propagation of the malady.

11. Ever bovine animal offered for sale ought to be accompanied by a certificate of health testifying that no epizootic has existed for at least six weeks in the commune from which it came.

12. In certain special cases to be determined by the veterinary official there may be prescribed the suspension of fairs and markets, the prohibition of the importation of animals from a suspected country, quarantine, the posting of notices at the entrance of infected circles or farms, and the writing of handbills and instructions warning the population of their obligations and of the precautions to be taken to prevent the appearance or extension of the malady.

13. An active supervision should be exercised, not only over the quarantined beasts, but also: *a*. Of animals exposed for sale in markets, fields, and fairs. *b*. Over animals lodged temporarily in the stables of inns adjoining the markets. *c*. In stables containing many animals subject to frequent changes, and when the malady has already made one or several outbreaks.

14. The duration of quarantine should be for 45 days at least after the disappearance of the disease.

15. At the release from quarantine the cattle should receive a second mark to annul the effect of the first.

16. The flesh of an animal slaughtered should not be used for food unless authorized by the veterinarian making the autopsy.

17. The skin should not be utilized until it has been steeped for at least twenty-four hours in an approved disinfectant solution.

18. Carcasses and cadavric debris unfit for food should be buried or so treated as to become absolutely inoffensive.

19. Stables, fair grounds, markets, and vehicles which have been occupied by diseased or suspected animals, should be carefully cleansed, disinfected, and purified. These differe t operations should be under the direction of a veterinarian.

20. A stable perfectly disinfected and purified by eight days' free ventilation may be refilled without danger.

21. Pasturages that have been occupied by diseased cattle should be shut up for forty days at least.

22. The different materials, objects, and instruments that have been employed in the slaughter, transportation, and burial of diseased or suspected animals should be destroyed or thoroughly disinfected. Forage and litter should be utilized for horses and other solipedes.

23. Persons who have become soiled by infecting materials should wash their hands, brush or wash their clothes, and wash their boots with a disinfectant solution.

24. All persons and animals capable of carrying the virus should be, as far as possible, kept from diseased animals, their carcasses, and cadavric débris.

25. Indemnity should be accorded: 1. For animals slaughtered officially. 2. For those that have died from inoculation. 3. For different objects or instruments of which the destruction is judged necessary.

26. Very heavy penalties should be imposed on persons who violate the different sanitary regulations ordered by the authorities.

27. A good organization of the veterinary service is the best guarantee of the application of the different measures prescribed.

28. A last and potent measure for securing the extinction of contagious pleuro-pneumonia consists in adopting a provision for the contagious diseases of animals as has been done for the phylloxera of the vine; to formulate an international agreement, in which shall be indicated the essential elements which ought to form the basis of the legislation to be adopted by each country which shall join it.

Degive justly accorded to lung plague a principal place among the plagues which are most detrimental to agriculture and the public wealth. In enumerating its anatomical lesions he lays stress on the inflammatory action being especially provocative of exudation into the interstitial connective tissues of the lung—interlobular, perivascular, interalveolar, and subpleural—upon the prominent implication of the lymphatics, upon the great extent of lungs involved even when the general symptoms of illness have only just appeared, upon the presence of lesions of different ages, implying a long standing and a slow and occult progress of the disease, and the existence in the exudate of the specific micrococcus of Willems, Bruylant, and Verriest. (Strangely enough, he fails to emphasize the infarctions and encysted sequestra which are so characteristic of the affection.) Under the head of physiological characters, beside the general symptoms of fever and inflammation of the lungs, he lays special stress on the two types of the disease, rapid and slow, the latter remaining insidious, hidden, and apart from indications furnished by auscultation and percussion, hardly recognizable throughout its entire course; on the mortality averaging 30 per.cent.; on the infectious property, very variable in different cases, but always highly conclusive when well marked. He seeks to establish a theory of spontaneity from the facts that in many cases contagion cannot be traced, that Grawitz, Greenfield, and Buchner have cultivated pathogenic fungi and bacteria until they have become harmless, and harmless germs until they have become pathogenic.

Under prophylaxis Degive adduces instances in which a thorough attention to the laws of hygiene in ventilation, lighting, feeding, &c., have seemed to arrest the propagation of the poison. In this as in the question of spontaneity the experience of Degive, limited to a country in which the disease constantly prevails, is misleading. He fails to take into account such sweeping evidence as the entire absence of the lung plague from America, South Africa, and Australasia until the occurrence of a single importation of disease, and its deadly prevalence in all three from that moment onward, notwithstanding that in the two last-named places the victims enjoyed an open-air life in mild and equable climate, the most favorable possible for the lungs. He further ignores for the instant the irregular and occult cases of the disease which confer immunity, and at once explains the sudden disappearance of the disease in particular herds coincidently with a better hygiene or a worse one, and the introduction of infection into a new locality where the best efforts of the veterinarians have failed to trace its source.

Degive strongly advocates inoculation, supporting his view by the following facts:

1. Out of 6,706 inoculated cattle placed in the same pathogenic conditions as 2,453 non-inoculated ones, 182 only, or about 2.71 per cent. among the first, and 660, or 26.90 per cent. among the second have contracted lung plague.

2. Out of 68 previously inoculated in the tail or by intravenous injection, and in which a second inoculation was practiced in a region rich in connective tissue (a deadly region), 61 have shown no local reaction, and 5 presented a slight inflammatory swelling, which in 6 beasts inoculated for the first time in the same dangerous regions, as test cases, all had an extensive inflammatory engorgement ending in death. Out of 6 animals inoculated by intravenous injection in the jugular and afterward subjected to 17 inoculations in the space of 16 months, 4 proved unaffected, 13 had slight inflammatory reaction, and 1 only had a considerable engorgement which did not prove fatal.

The immunity secured by inoculation has lasted four and five years, as observed by Ziegenbein, in animals constantly exposed to infection, and for one year in 16 test cases in the experiments of the Central Society of Veterinary Medicine of Paris.

The drawbacks to the operation are:

1. All the inoculated do not acquire a perfect immunity.
2. Inoculation preserves and spreads the poison.
3. A certain small proportion die from the extensive swellings and gangrene consequent on the inoculation.

That all the inoculated are not protected is shown in the above statistics; some highly susceptible animals still contract the disease as some men contract small-pox after vaccination. To secure a better immunity it is advised to make more careful selection of the virus from a lung engorged yellow and already slightly fibrinous, or from the subcutaneous connective tissue in an inoculated region rich in that tissue, to repeat the operation after a certain lapse of time, as advised by Willems, and with weakened virus to inoculate in a region rich in connective tissue. This, it is claimed, can be done safely with virus which has been kept six weeks in a hermetically sealed tube (Pasteur), or with what has been cultivated for a length of time in flasks in a special manner (Bruylants and Verriest). Virus diluted with 50, 100, and 500 times its amount of inert liquid still remained dangerous. (Vandermies: Central Society of Veterinary Medicine, Paris.)

The danger of the propagation of the disease by inoculation is really very slight. Yet he acknowledges the presence of the virulent micrococcus in the inoculation exudate, and advocates inoculation as a means of procuring pure virus for further *protective* inoculation.

To obviate the slight danger of lung infection through inoculation he advocates the use of attenuated virus (*à la* Pasteur, or *à les* Bruylants *et* Verriest) and the intravenous injection of the virus pure or attenuated. (It is to be noted that an *absolute immunity* is not claimed for any method, nor an *absolute protection against the propagation of the disease by the inoculated;* the claim is that these may be reduced to a very small figure. It follows that the protective inoculation is a measure of *repression, not extinction*, and to a case like ours, where a prompt *stamping out* is imperative, it is quite inapplicable.) This is the more evident from Degive's opinion that "all inoculated animals are to be regarded as suspected of infection, and treated as such, and that after the completion of the operation all virulent products should be thoroughly destroyed or buried."

To obviate losses from inoculation he particularly advises the use of *attenuated virus* or intravenous injection, and to avoid the operation during hot and rainy seasons.

For the *destruction of the poison* it is necessary to attend to all fodder (which has remained infecting for 3, 4, and even 9 months), all articles about the stables, or suspected cattle, all other animals (not bovine) that have cohabited with them, all places (buildings, &c.) where they have been, all vehicles used for their conveyance or that of their products, and all persons that have come near them or the infected places. The infected herd, the carcasses, and fresh products must receive, of course, the first attention. Thorough destruction or disinfection of all these is absolutely essential.

As the discovery of infection is the corner-stone of success, it must be made obligatory on all owners and attendants on cattle, on all veterinarians and inspectors of abattoirs and meat markets, to promptly report all cases of the disease, under a heavy penalty for disobedience and for the owner a liberal indemnity for cattle and objects destroyed.

At his *visit* the official veterinarian must note the animals sick and exposed, the source of the infection, what objects have become infected, what persons and animals may become vehicles of the virus, and what measures (especially isolation and killing) are required.

Inspection of fairs, markets, and herds in an infected district, and visit, every fortnight, of large stables, then having frequent changes, and those those that have been infected, are requisite.

Slaughter should be resorted to for the sick and all suspected of disease, no treatment being permitted. Recovered animals should in all cases be used for meat only. All slaughter of subjects out of an infected herd should be in the presence of as few persons as possible and under the inspection of a veterinarian. The animals suspected of being infected should be inoculated. (The provision against treatment should also preclude inoculation if extinction of the disease is desired.)

For indemnity as a means of securing early reports he advocates, at least one-half the sound value for beasts suspected, yet fit for human consumption, and at least three-fourths the sound value for those that are diseased and unfit for food. Better still to completely extirpate the infected herd and indemnify to the extent of the full value, as in Holland and England; the state to retain in such a case all proceeds from flesh and hide.

Under *isolation* come: (*a*) *Sequestration* in a particular place near which no other cattle nor other animals are allowed.

(*b*) *Cantonnement or parkage* in a secluded place inclosed by walls, hedges, barriers, palisades or water, and safely apart from roads, parks, &c., frequented by other stock.

(*c*) *Sanitary zones or circles*, including a part or whole of a village or commune or several communes, separated from all communication with outside districts.

(*d*) *Census and marking* are essential to a perfect control of such secluded places.

(*e*) *Prohibition of movement* of all sick and suspected animals is absolutely essential. Under special precautions fat cattle may be moved in closed wagons to slaughter, and work-oxen may be utilized on certain prescribed fields if there is no danger of communication with other cattle, direct or indirect; no cattle should be exposed for sale anywhere without an official certificate that no epizootic has existed for six weeks or more in the commune from which they were drawn.

(*f*) *Suspension of fairs and markets* is only demanded when the lung plague has attained to an extensive prevalence.

(*g*) *Importation of cattle should be interdicted* from any infected country unless they are accompanied by a certificate of health dated six days before and showing that no lung-plague has existed for at least six weeks in the place from which they came. (Such provisions show the aim at restriction rather than extinction of the malady, as they could not arrest the occult cases nor those having a long period of incubation.—J. L.)

(*h*) *Quarantine* of newly-bought animals for five or six weeks is an important precaution.

(*i*) *Posting notices of infection* at the entrance of infected zones or farms.

(*j*) *Handbills and instructions* for the people in or near the infected area.

(*k*) *Surveillance* with very frequent visitation by inspectors and police.

Preventive medication for animals exposed to infection may embrace *setons medicated with* hellebore, and *antiseptics*, such as ferric sulphate, alkaline sulphites, carbolic acid, borax, tar, chlorine.

All restrictions may be removed forty days after the last case of disease has been disposed of and the place and objects disinfected. (This is entirely inconsistent with what he has already advanced as to occult cases, chronic cases, and long periods of incubation which could so easily exceed twice the forty days required. It is another indication of an aim at restriction rather than extinction.)—J. L.

If *carcasses* have to be removed, this should be done in wagons with close joints, so that nothing shall escape, and drawn by horses under police supervision. The carcass and diseased products may be deeply buried, burned, rendered, or dissolved in sulphuric acid. It is often utilized for food, but this should be prohibited when the lung lesions are very extensive, advanced, and complicated by gangrene, when there is ulceration of the bowels, or when the flesh is flaccid, decolorized, ecchymosed, or the seat of serous infiltration. The *skin* may be sent to the tannery after steeping a length of time in milk of lime, or solution of chloride of lime, or zinc, or carbolic acid.

Disinfection of stables demands washing, scraping, and the thorough application of liquid disinfectants. Manure, fodder, and litter should be burned or disinfected, or the latter may be fed to horses. Horses and other animals that have been with diseased cattle should have their surface cleaned and disinfected by an antiseptic solution.

LEBLANC'S VIEWS.

Leblanc sought to throw doubt on the diagnosis of lung plague during life, on the specific character of the lesions seen in the carcass, and on the value of inoculation. Many inoculated animals have already had the disease and are protected by that; cases already infected before inoculation have the disease aggravated by the operation; the poison, and therefore the disease, is preserved and perpetuated in certain districts by the practice of inoculation; inoculation is no certain prophylactic, for just as in the same lung we see chronic lesions side by side with the acute, implying a second attack, so may an attack follow a successful inoculation; and finally, the same measures of segregation and slaughter usually employed with inoculation would be successful without that operation.

PUTZ'S VIEWS.

Putz does not hesitate to pronounce the physiological symptoms and pathological lesions as together reasonably pathognomic. He is a strong partisan of inoculation, provided the operation is repeated after a lapse of time, and above all if repeated again and again. It is useless or injurious for animals that are already infected, and the duration of the immunity acquired in successful cases varies with the individual susceptibility, as does vaccination for the prevention of small pox. It is especially valuable in places where movements of great numbers of cattle are continually going on, and where sequestration is in consequence impossible. Where, on the other hand, there are few movements of stock the isolation and slaughter of the diseased and strongly suspected beasts is speedily effective. In Holland with inoculation the disease has been confined to the narrowest limits, while in most parts of Germany it has been stationary or increasing, and in Saxony with little inoculation it has in recent years attacked three times the number of victims seized in 1875–'76.

In addition to the measures of sequestration he advocates: 1. That the quarantine mark should bear the year so that it may be afterward known when they were diseased or suspected. 2. That all diseased and strongly suspected animals should be killed, and that the entire herd should be slaughtered when judged necessary, indemnity being granted for the same. 3. When, owing to frequent changes in a large herd, or when, from economical considerations such herd may not be slaughtered, the same should be compulsorily inoculated. Inoculation may be authorized in an infected country, if desired, by the proprietor, and consequent losses should be paid for. 4. Every beast from a quarantined herd which dies or is killed ought to be the subject of an autopsy by a competent person. With this precaution such cattle should be devoted to slaughter for beef as much as possible, no necessary precaution being forgotten.

ACTION OF THE CONGRESS.

A.—DIAGNOSIS.

The question of *diagnosis* of lung plague gave rise to a somewhat lengthy, animated, and fruitless discussion as to whether this disease *can arise spontaneously*, or whether *it is everywhere and always the result of contagion*. The advocates of a spontaneous origin of the disease acknowledged that the occurrence of a spontaneous case was extremely rare, and that in spite of spontaneous cases a system of repression, based on a constant assumption of contagion, was the best; yet they claimed, as already stated of Degive, that the malady may originate by the transformation of harmless germs into virulent ones, or it may be by the transformation of normal histological elements of the body into diseased elements having a power of propagating themselves indefinitely. The opponents, on the other hand, held that the non-appearance of this disease in historic time, in any country in which it had not previously existed, unless in cases where its introduction could be clearly traced to the importation of a diseased animal or its products, and its continued absence from all countries into which no such importation had been made implied, unequivocally, that the assumed cases of spontaneity were also cases of infection, though investigation had failed to show the precise channel by which the germs had been introduced. A comparison of the nationalities of the advocates of spontaneity and against it is very instructive as showing that the believers in spontaneity are those whose experience has been gained at the termini of the cattle traffic from Central and Eastern Europe, at points (Belgium, France), in short, where the infection of lung plague is being constantly imported, and from which it is never entirely absent, whereas the disbelievers in spontaneity are mainly from countries (England, Sweden, Switzerland, Roumania, America, &c.), in which lung plague has been stamped out, or into which it has been first introduced in recent times by a well-attested importation of disease, and where its area of prevalence is sharply limited to places infected through such importations.

This well illustrates the predominating influence of the immediate surroundings. Had the able advocates of spontaneity lived in Spain or Portugal, where herds abound, but to which the lung plague has never penetrated, or in Scandinavia, where its occasional importations have been as persistently stamped out; or in Switzerland, the immemorial home of the plague, but from which it has been expelled; or in England, which it respected until it was imported in 1839 and where it has prevailed ever since, but still spares the exclusively breeding districts; or in the United States, where it was imported in 1848 and 1859, and where it was effectually stamped out in the fenced farms of New England but continues to prevail through the constant changes and successive infections in the city dairies of the Middle Atlantic States, and finally where the whole West and South maintains a perfect immunity; or in South Africa or Australasia, where the disease, long unknown, has spread from single importations and from the constant mingling of herds maintains an universal prevalence; or, finally, in Canada, Newfoundland, Mexico, or South America, to which the plague has not yet been imported and where, as in the exclusively breeding districts of infected countries, no *spontaneous* case has ever occurred to start it on its desolating career, they would have realized that they were advocating a mere phantom danger and that the plague which has failed to appear in historic time in a country not already infected from without may safely be trusted not to appear in the future with such exotic contagion. They would no more argue from the first case of the plague than the botanist would argue from the first oak that oaks must now appear without seed or slips; they would accept the unvarying testimony from all parts of the inhabited earth which are not yet infected or which have been infected in historic time that every extension of this plague has been by contagion and by contagion alone.

On motion of Wirz the following was adopted as the first resolution:

1. From an anatomical point of view, at least in its relation to veterinary police, every pneumonia (of cattle) which is lobular and at the same time interlobular, and the development of which does not depend on traumatic local causes, should be considered as epizootic contagious pleuro-pneumonia.

The second resolution was modified by adding the word *contagious*, so that it might read:

2. From a physiological stand-point, epizootic contagious pleuro-pneumonia is specially characterized, in the living animal, by the contagious character and by the symptoms of lobar-pneumonia.

To the third resolution the following wording was given:

3. There ought to be considered as—

(*a*) Suspected of epizootic contagious pleuro-pneumonia every animal which in an infected place manifests symptoms of fever or of disease of the chest.

(*b*) Suspected of contamination every animal found in an infected stable, or which has been in one within three months, or which has been exposed to infection in any other way.

The fourth resolution was suppressed on motion of Lydtin and Wirz.

B.—PROPHYLAXIS.

On motion of Zundel and Lydtin the congress decided to take up first the question of stamping out.

On motion of the same, Article 2 was modified and finally passed so as to read as follows:

2. Recognizing that from the point of view of sanitary police epizootic pleuro-pneumonia propagates itself only by contagion, and is usually incurable and fatal, this congress declares that animals affected by the malady or suspected of it should be sacrificed as quickly as possible.

The third article was adopted with the single change of substituting contaminated for suspected of contamination or very much exposed to contagion, the word retaining the idea of exposed to contagion. It read thus:

3. Contaminated animals should be isolated or sacrificed. The slaughter of contaminated animals is especially indicated when the disease manifests itself very exceptionally or for the first time in a stable belonging to a commune rich in cattle.

On the question of the value of inoculation as a preventive, much discussion ensued, the great majority, however, according to it the power of protection to a certain extent. A number, however, of these last, and especially those who like Berdez (Switzerland), Law (New York), and others had had a favorable experience of stamping out, deprecated inoculation in any country where it was possible to promptly extirpate the plague by the radical measures of slaughter and disinfection. Wirz reported that of the 182,308 cattle inoculated in Holland, in the past four years (1878–1882) the losses from the operation had been under 1 per cent., and the protection had been satisfactory. Law repeated the inoculation of 10 cattle with sterilized virus which obivated the danger of infection from the inoculated, and which, in a six-month's test, by inoculation with attested virulent lymph, and by residence in infected herds, had proved perfectly satisfactory. Willem's proposition that "all scientific interpretations being reserved no fact in practice has proved the contamination of a healthy by an inoculated animal" was voted down, and the following of Potteral adopted:

There is no proof that an inoculated animal cannot transmit the disease to a healthy one.

The following, moved by Bouley, was adopted:

3. We have to-day experimental proof that it is possible to invest the organism of animals of the bovine species with an immunity from contagious pleuro-pneumonia by inoculation with the virus of this malady.

Two applications of inoculation were recognized—preventive inoculation (inoculation in the absence of the disease), and inoculation of necessity, (inoculation of animals dangerously exposed to infection). As an amendment to Article 4 the following was passed:

4. Preventive inoculation, that is to say, that which is practiced where the malady does not prevail in a country, ought to be absolutely rejected; inoculation, so called, of necessity, that is to say, that which is practiced when the malady exists in a herd, may be permitted, but not made obligatory.

On the motion of Wirz, Article 5 was stricken out; Article 6 was modified so as to read:

6. The inoculation should always be done by a veterinarian.

Article 7 was dropped.

On Degive's motion Article 8 was altered to—

8. Inoculated animals should be reported to the authorities.

Articles 9 to 13, inclusive, were not called in question. On motion of Müller the following was adopted:

14. The duration of quarantine should be six months at least after the disappearance of the disease.

Articles 15 to 19, inclusive, were not objected to.

On motion of Lydtin, Müller, and Potteral, Article 20 was altered as follows:

20. A stable should not be used again for animals until it has been completely evacuated and properly disinfected, and then purified by eight days' exposure to free ventilation.

On motion of Bouley and Aune, Article 21 was altered as follows:

21. Pastures that have been frequented by diseased animals ought to be quarantined for at least fifteen days.

Articles 22 to 24, inclusive, were passed.

On motion of Quivogne, Potteral, and Lydtin, Article 25 was modified as follows:

> 25. It is proper to grant an indemnity to owners for animals sacrificed by official order, and for the expense of disinfection. The indemnity should amount to four-fifths of the value of the animals; and to the full value, deduction being made of the value of portions of the carcass that can be utilized if the animal should prove healthy.

Articles 26 to 28 were adopted without question.

REMARKS.

Without seeking to detract from the importance of any one position taken by the congress, it may still be well to emphasize some that have a special value to the American statesman.

1. The extension of the incubation of lung-plague for months, and the frequent occurrence of occult and unrecognized cases of the disease, show how essential it is to stop all movement of animals in infected districts, except under license, after an extended supervision, including statistics constantly corrected; also to kill out an entire infected herd, or to maintain the above-named strict supervision for a long period (six months); also, to prohibit the contact of adjacent herds in neighboring parks, &c., and their successive presence in the same pastures, on the same roads, or at the same drinking troughs. These dangers are just those of which it has been most difficult to persuade our United States legislators and our non-veterinary administrators of State sanitary laws, and on the fundamental blunders made on these points depend our failure hitherto to extirpate lung-plague.

2. The idea of the spontaneous origin of lung-plague in the present day is effectually set aside. An abiding confidence in our perfect security from this disease apart from imported virus is essential to thorough work. Whenever the possibility of spontaneous cases is admitted this will be made a cloak for slovenly and ineffective work.

3. The voice of the representative veterinarians of Europe and America has been given against the assumption that inoculated animals cannot infect a sound animal. This is a decision of no small importance, as this operation of inoculation is extensively practiced among us, and though it enables the individual owner when left to his own resources to save the great body of his herd, yet when the state undertakes to stamp out the plague, its practice becomes a serious hindrance by increasing and diffusing the virus.

4. In spite of the difficulty or impossibility of controlling the enormous cattle traffic which is constantly flowing westward through the center of Europe, and the consequent temptation to adopt measures of *repression* and *restriction* rather than those of *extinction*, the congress declares strongly in favor of the instant slaughter of all diseased animals and of those suspected of disease. How much more should we who have to deal with but a mere patch of inspection relatively to our territory promptly destroy every animal and every herd in which infection is found?

5. Even in Europe the veterinary sanitary authorities feel that action by isolated states aiming at the suppression or extinction of lung-plague is wofully ineffective, and they demand that the veterinary sanitary police administration shall overstep the national boundaries and be made continental, to the extent that the different nations shall agree upon a uniform law, under which the disease shall be promptly stamped out or honorably and effectually shut up in any district where it may appear,

so that an official certificate may give an absolute guarantee of soundness. How much more should the United States, bound into one nation and having one common Federal legislative body, and one Federal administration, arrange for a single law on this subject for all the States and Territories and for its uniform administration, whether through State or national officials? Hitherto the varying laws in different States have been a source of constant uncertainty, trouble, and loss to dealers, and yet no certain guarantee against the extension of the plague from State to State.

6. In dealing with an insidious affection like lung-plague the veterinary profession in Europe realize the necessity of adopting every means calculated to secure information of outbreaks, and the restriction of surreptitious movements of animals; they accordingly declare in favor of indemnity to four-fifths of the sound value for sick cattle sacrificed and the full value for cattle exposed to infection but not yet diseased, and also for heavy penalties for all failures to comply with the law. In our own State of Pennsylvania, infected herds have been taken possession of by the State, and all that subsequently sickened have been paid for at full appraised value, with results incomparably better than where the law has been to give small indemnities and impose large penalties. Our legislators should realize from this combined experience of Europe and America that niggardly indemnities mean concealment, smuggling, and extension of the disease, while with liberal remuneration for the cattle taken the disease may be stamped out at a mere fraction of the outlay that would otherwise be necessary. In city dairies, where most of our lung-plague is to be found, the full value of the animals killed is far from compensating the owner for the interruption of his business until his herd and stables can be pronounced sound. No economy is more false than that which saves on the payment for infected cattle slaughtered at the expense of concealment and surreptitious diffusion of the disease.

3. EDUCATION IN VETERINARY MEDICINE.

This subject was introduced by two separate reports—one by Hugues, of Brussels, the other by Professor Wirz, of Utrecht, and Müller, of Berlin.

HUGUES' REPORT.

A.—Education in veterinary medicine and the social position of the veterinarian demand a thorough preliminary instruction corresponding to the classes in humanity or the complete professional ones.

B.—Education in veterinary medicine should be theoretical, scientific, practical, experimental, and educational, in giving to each of these the relative importance which the real needs of professional work demands.

To this end we ask—

1. That the studies should extend over five years.
2. That the exterior of domestic animals be made the subject of an essentially practical course, of which the study of animal mechanics shall be the basis.
3. That there ought to be created in every school a course of equitation.
4. That exercises at the forge should be abolished.
5. That the course of special pathology should be abolished.
6. That there should be theoretical and practical instruction in the inspection of alimentary matters of animal origin.
7. That there should be at least two professors of clinics in each school.
8. That a residence outside the walls (external) should be obligatory at least for the last two years of study.
9. That a period of probation (stage) be imposed as complementary to the school studies.

10. That practitioners should be made part of the examining boards.
11. That the appointment of professors should be made on the double basis of *concours* and of scientific reputation; that assistants or tutors should be nominated on the proposition of the professional college.

While we cannot follow Hugues through his elaborate report, yet we may cull a few of the points made in favor of his propositions.

A liberal profession is marked by solidarity; it is cosmopolitan, knows no territorial frontier, no nationality; it is the product of civilization, and protected by universal science. The liberal profession of medicine is one; its methods only differ according to the species to which it is applied. The two professions of medicine—human and veterinary—are sisters, equally liberal, and demand an equally extended preliminary training and give an equal right to consideration. To secure this equality the education for the one must be as thorough as for the other, alike in its literary, scientific, and special features.

Continental Europe has thirty-three well-equipped veterinary schools, each a Government institution, controlled and supported by the state. Great Britain has four veterinary schools, none of which is under state support nor control aside from the charters under which they are maintained. In England and Austria the course of study extends over three years; in Germany and Switzerland, three years and a half; in France, Belgium, Holland, Denmark, Sweden, Russia, and Italy, four years; and in Roumania, Spain, and Portugal, five years.

As an example of the curriculum, that of the Brussels school will alone be given.

VETERINARY SCHOOL OF BRUSSELS.

FIRST YEAR—WINTER SEMESTER.

Descriptive anatomy, 1½ hour per week.
Dissections, 9 hours.
Tuition in chemistry or physics, 1½ hours.
Lectures in chemistry or physics, 4½ hours.
Botany, 3 hours.
Examination in chemistry or physics, 1½ hour.

SUMMER SEMESTER.

Botany, 4½ hours.
Botanical excursion.
Tuition in botany, 1½ hour.
Chemistry or physics, 4½ hours.
Tuition in chemistry or physics, 1½ hour.
Zoology, 3 hours.
Tuition in descriptive anatomy, 1½ hour daily.
Examination in chemistry or physics, 1½ hour.

SECOND YEAR—WINTER SEMESTER.

Tuition in chemistry, 1½ hour.
Tuition in physics, 1½ hour.
Examination in chemistry or physics, 1½ hour.
General anatomy and physiology, 4½ hours.
Tuition, general anatomy and physiology, 1½ hour.
Descriptive anatomy, 3 hours.
Dissections or exercises in histology, 4½ hours.
Dissections, 15 hours.
Physics or chemistry, 4½ hours.
Comparative anatomy, 1¾ hour.

SUMMER SEMESTER.

Tuition in physics or chemistry, 1½ hour.
Tuition in chemistry, 1½ hour.
Tuition in general anatomy or physiology, 1½ hour.
Work in histology, 4½ hours.
Physics or chemistry, 4½ hours.
General anatomy and physiology, 4½ hours.
Comparative anatomy, 1¾ hour.
Tuition in comparative anatomy, 1½ hour every 15 days.
Work at the forge (*maréchalerie*) 3 hours.
Examination on chemistry or physics, 1½ hour.

THIRD YEAR—WINTER SEMESTER.

Clinics, 2 hours daily.
Pharmacology, 3 hours weekly.
Tuition in general anatomy and physiology, 1½ hour.
Theory of shoeing, 1½ hour.
General pathology, and special and pathological anatomy, 4½ hours.
Tuition in general and special pathology, and pathological anatomy, 1½ hour.
Exercises in operative medicine (surgery), 3 hours.
Exterior (form), 3 hours.
Tuition on exterior, 1½ hour every 15 days.
Special therapeutics and pharmaco-dynamics, 3 hours.
Topographical anatomy, 1½ hour.
Work at forge, 1½ hour.

SUMMER SEMESTER.

Clinics, 2 hours daily.
Operations on the foot, 1½ hour per week.
Operative medicine, 4½ hours.
Demonstrations in pathological anatomy, 1½ hour.
General pathology and special and pathological anatomy, 4½ hours.
Tuition in clinics, 1½ hour.
Pharmacology, 1½ hour.
General therapeutics and pharmaco-dynamics, 1½ hour.
Pharmaceutical manipulations, 1½ hour.
Tuition in general pathology and special pathological anatomy, 1½ hour.
Zootechny, 1 hour.

FOURTH YEAR—WINTER SEMESTER.

Clinics, 2 hours daily.
Tuition, clinical, 1½ hour per week.
Surgical pathology, 4½ hours.
Pharmaceutical manipulations, 3 hours.
Obstetrics, 1½ hour.
Practical operative medicine, 3 hours.
Work at forge, 3 hours.
Zootechny, 1½ hour.
Tuition in surgical pathology, 1½ hour.
Constitutional law, 1½ hour.
Equitation, 4 hours.

SUMMER SEMESTER.

Clinics, 2 hours daily (2 hours weekly clinics in chair).
Work at forge, 3½ hours.
Pharmaceutical manipulations, 3 hours.
Tuition, clinical, 1½ hour.
Medical jurisprudence and sanitary police, 1½ hour.
Tuition in surgical pathology, 1½ hour.
Zootechny, 3 hours.
Examination of meats, 1½ hour.
Zootechnic conferences, 1 hour.
Tuition in zootechny, 1½ hour.
Equitation, 4 hours.
Tuition in medical jurisprudence and sanitary police, 1½ hour.
Constitutional law, 1½ hour.

Others of the schools, and notably those of France, give instruction in modern languages and literature, which Hugues holds should be obtained before entering the veterinary school. To further relieve the curriculum and give more time to the exclusively professional studies, he would abolish the class on constitutional law, that on shoeing, and even that on special pathology as taught from the chair, thus throwing the student back on books and clinical teaching for instruction in the practice of medicine. The need of some relief is well illustrated in the fact that a large proportion of students to-day exceed the allotted period of study preliminary to taking a degree. Thus at Brussels in former times a failure to pass in four years was altogether exceptional, while under the modern crowding of studies but 33 out of 77 students have passed in this prescribed period; of the remaining 44 students 20 took five years, 20 six years, 1 seven years, and 3 eight years.

The curriculum has greatly outstripped the ability of the student to cope with it, and the two should be adjusted so that the majority may be able to graduate in the prescribed period. Though something may be done in eliminating subjects that are not purely professional, yet, with the rapid advancement of science, the exclusively professional work incumbent on the student tends constantly to increase, and the strain must be met by securing a better preliminary training, and by extending the curriculum to five years. Thus, for admission to the school, a knowledge of one or more modern languages should be demanded; those of the adjoining countries being always valuable as giving a key to their literature and as being essential in the administration of sanitary police. The rudiments of Latin are very useful, but not indispensable. National and foreign literature have no occasion to appear in the curriculum, and should not be a condition of entrance. So of mathematics and the natural sciences. If the same could be applied to physics and chemistry it would be well, but this would be asking too much of a boy of seventeen, and would endanger superficiality in all.

Then if the curriculum were extended to five years, the present double examination might be profitably extended to three; the first in sciences, the second as candidate in veterinary medicine, on anatomy, physiology, histology, physics, chemistry, and perhaps the exterior, and the third one, pathological biology, therapeutics, surgery, clinics, hygiene, zootechnnics, sanitary police, &c.

The examining boards should be composed of the faculty of the school and a certain proportion of veterinary practitioners. This will tend to correct any tendency in the schools to a too exclusive attention to scientific minutiæ at the expense of the even more important matters of daily practice, and give a special value and guarantee to the examination and diploma. On the other hand, the intimate knowledge of the candidate on the part of the professor will enable the board to qualify the results of a hurried examination by the record of five years of continuous work.

In the appointment of teachers much is required. The professor should be a man of high morality, one inspiring respect and esteem, who knows his subject thoroughly, and who yet can condense it to the demands of the case, and present it clearly, plainly, and concisely, yet in such a way as to engage the enthusiasm of his students. He may be a good professor without being a *savant*, and he may be a real *savant* yet a very poor professor. To know and to teach are different things. A mind quick to write, subtle in the analysis of facts, facile in the assimilation of all contemporary progress, a ripe judgment, the power to present the analysis or synthesis in the form of a clear attractive résumé; these are the essential qualities, the rôle and mission of the professor. He must besides have such a knowledge of the entire curriculum as will enable him to direct his work parallel to that of his colleagues without exposing himself to contradiction, or unnecessary repetition by another chair. To successful teaching there must be a unity and harmony in the entire field of work. This necessitates that every professor in a chair bearing directly on specific veterinary instruction should be himself a veterinarian.

In the appointment of professors regard should be had to the aptitude for scientific work already shown by the candidate, and the aptitude to teach, as shown experimentally before a *concours*. The *concours* alone in which the candidate is made to exhibit his teaching powers practically may often select the fluent but superficial man and reject the real scientist, as it has actually at different times rejected the illustrious Bichat, the creator of microscopical anatomy, Claude Bernard, the founder of general physiology, and Dupin, the learned procurator-general of the court of causation. But, as corrected by reputation for work done outside the *concours* the latter becomes a means of the highest value in selecting a man who joins superior didactic power to a profound scientific knowledge and acumen.

Tutors should only be chosen from graduates, in which case the excellence in examination should coincide with the judgment of the professor in charge of the department in making the selection. When professors are recruited from the ranks of the tutors they should have the experience of several years' practice.

REPORT BY PROFESSORS MÜLLER AND WIRZ.

Müller and Wirz indorse in the main the principles enunciated by the International Congress at Zurich in 1867, but propose certain modifications. The Zurich resolutions were as follows:

1. The preparatory studies should be as extended for veterinary medicine as for human medicine. It is desirable that we should, as far as possible, demand for entrance to the veterinary schools the same knowledge as for the university studies.

Since, for various reasons, we are not yet able to enforce such a rule, the congress is of opinion that the minimum knowledge preparatory to the special studies of veterinary medicine should correspond to those acquired in the second (class) of a school preparatory to university studies.

Those who have not pursued the course of such an institution should not be admitted to the special veterinary studies until they prove that they are possessed of an education corresponding to that of the class above named.

2. Three years of special studies at least are necessary to secure the rank of veterinarian.

There is no call for the creation of veterinarians of different classes based on differences in the degree of instruction.

3. Veterinary schools may be separate, independent establishments, or joined to by universities or other institutions of higher learning, but their instruction should be special chairs. We cannot but condemn the arrangement in which a single professor is charged with the duty of educating veterinarians, this mode of teaching being absolutely insufficient.

4. An organization of the instruction conformed to the above principles should be adopted above all when the practice of veterinary medicine is well established and regulated.

PROPOSITIONS OF MÜLLER AND WIRZ.

I.

1. The preparatory studies demanded for the study of veterinary medicine ought to be the same as those exacted of the student of human medicine.

2. As for various reasons this first principle cannot yet be enforced, we ought at least to require that all who wish to enter on the study of veterinary medicine should *at least* possess the acquirements necessary to admit them to the highest classes of a superior school giving an ordinary good education.

As an institution giving an ordinary good education, we mean those schools which give a right to students that have completed their course to admission to the higher or university studies. (Gymnasiums, lyceums, atheneès, Latin schools, colleges), (and superior professional schools, *Realschulen, erster ordnung*, of Germany), (Wirz and Müller), in which Latin is obligatory (Müller).

3. Candidates unprovided with a certificate of admission to the first class of one of these schools should show by a special examination that they are possessed of an equivalent education.

II.

There is no call for the creation of veterinarians of different classes having a different amount of preparatory and veterinary education.

III.

1. Four years' study, at least, are requisite to make a full study of veterinary medicine, if that is made to embrace physical and natural sciences.

2. The instruction of the first two years (four first semesters) should embrace the following branches: physics, chemistry, natural history (geology, mineralogy, botany, and zoology), anatomy, histology, physiology, and shoeing, with the practical work

attaching to them. A course of practice and demonstrations in micrography should always be included.

3. In the same period may be taught the zootechnic branches, comprehending the natural history of domestic animals, the exterior, and zootechny proper.

4. Clinical teaching should continue through the whole of the last two years of study. That the practical instruction of the students may be complete it is absolutely necessary to have beside a stationary and consulting clinic (hospital clinic and polyclinic), an ambulatory clinic (outside clinic).

5. Practical instruction in shoeing cannot be condemned as useless, but it ought to be limited to the end proposed; this instruction is on the whole very desirable.

6. The inspection of meats of the butchery is an absolutely essential branch of veterinary education.

IV.

1. At the end of the second year of study (fourth semester) the students ought to be examined on the branches they have studied during the two preceding years. None should enter on the studies of the third year until he has satisfactorily passed this examination (of candidate, or in physical and natural sciences).

2. None should be admitted to the examination for veterinarian until he has passed that of candidate in veterinary medicine.

(The examination in veterinary medicine should embrace only those branches which have not formed part of the candidature examination.—*Wirz.*)

(The examination in veterinary medicine should embrace, beside anatomy and physiology, all branches of instruction not included in the examination of the candidate.—*Müller.*)

3. The regulations for veterinary examinations should, as far as possible, be absolutely, or at least essentially, the same for all countries.

V.

1. The system of residence in the school (internal) is not the best for the pursuit of veterinary studies and the social education of veterinarians.

2. If peculiar circumstances, proper to any country, forbid the abolition of residence (internal), the students should at least be allowed entire liberty outside the prescribed course; the control of the internal ought to be as liberal as possible.

3. Obligatory "internal" should be abolished.

VI.

Veterinary schools may be independent establishments, or they may be connected with universities or institutions for the higher education; but veterinary medicine should have its special chairs. One cannot but disapprove of institutions in which all branches of veterinary education are divided in a very limited number of university chairs; such a system is absolutely insufficient.

VII.

1. Professors in veterinary schools should be possessors of veterinary diplomas; an exception to this rule may be admitted in the case of those teaching the preliminary courses of physics and natural sciences.

2. It is very desirable that veterinarians before being called to a professorship should have practiced veterinary medicine for some years.

3. The diploma of physician or M. D. should not in itself render the holder eligible to a veterinary professorship.

4. The professors ought to be selected by preference from among the assistants, and upon the proposition of the faculty of the school in question.

5. Finally, to be able always to complete the professional body, there ought to be created numerous places for assistants.

REASONS.

As the requirements for entering the university are more than can at present be enforced, and more than is always required for the study of medicine, for polytechnic schools, schools of mines, &c., they should not be required at present. The same conditions of admission should be maintained in all veterinary schools, and for all students, national or foreign, for all, indeed, who do not attend as simple auditors. The entrance examination should be made before a commission of the faculty who best know the acquirements requisite for the pursuit of the study.

Since the Zurich congress the great additions to veterinary studies in practical chemistry, micrography, &c., necessitates the extension of the course to four years at least. Wirz thinks even five years desirable.

Hugues' proposal to abolish the chair of special pathology is untenable. So long as the clinic does not furnish abundance of material to show and demonstrate to all the students cases of every disease it is impossible to agree to his proposal.

While recognizing how much veterinary medicine is indebted to human medicine, they cannot admit that the knowledge of the one fits for the teaching of the other. Medical professors are far from comprehending all the requirements of veterinary education, or the exigencies of veterinary practice. Most of them continue ignorant of these from lack of inclination as much as lack of opportunity, and the education suffers proportionally. For professors and assistants alike a veterinary diploma is a *sine qua non.*

ACTION OF THE CONGRESS.

After discussion the following was adopted as the first proposition:

1. For admission to veterinary studies one must be batchelor *ès lettres* or *ès sciences*, that is to say, he must have finished the studies of the secondary education.

The second proposition of Müller and Wirz was adopted, and reads:

2. There is no call for the creation of veterinarians of different classes, having a different amount of preparatory and veterinary education.

The third proposition was adopted with modifications of the second paragraph, so as to drop all reference to practical work, and of the fourth paragraph, so as to provide for two clinical professors in each school, and by the dropping of paragraphs 3 and 5. As altered, it reads thus:

3. Four years of study at least are requisite to make a full study of veterinary medicine, if that is made to embrace physics and natural sciences.

(*a*) The instruction of the two first years (four first semesters) should embrace the following branches: physics, chemistry, natural history (geology, mineralogy, botany, and zoology), anatomy, histoloy, physiology, and shoeing. A course of practice and demonstrations in micrography should always be included.

(*b*) Clinical teaching should continue through the whole of the last two years of study. That the practical instruction of the students may be complete it is absolutely necessary to have besides a stationary and consulting clinic (hospital clinic and polyclinic), an ambulatory clinic (outside clinic); there ought to be at least two professors of clinic.

(*c*) The inspection of meats of the butchery is an absolutely essential branch of veterinary education.

The fourth proposition was modified by adoption of a motion by Quivogne and Larmet to have yearly examinations, by one by Wehenkel to make a two years' clinical course obligatory, and by one by Eraers and Leblanc that examining boards should be composed of professors and practitioners. As adopted, it reads thus:

4. At the end of each year the veterinary students should be examined on the studies which they have been taught that year; no one should be allowed to follow the course of the advanced year without having passed this examination.

No one should be admitted to examination for the degree of veterinarian who has not followed a course of clinical instruction for two years after having passed the examination of the second year of study.

The board of examiners for conferring grades should always be formed partly of professors and partly of practitioners.

On motion of Quivogne proposition third was altered to:

5. "Internal" and "external" are optional in veterinary schools.

Proposition 6 was adopted unchanged, and on motion of Wirz, Fleming, and Laiutard, an expression in favor of the maintenance of all veterinary schools by the state. As adopted it reads:

6. Veterinary schools may be independent institutions, or they may be connected with universities or institutions for the higher education, but veterinary medicine should have its special chairs. One cannot but disapprove of the creation of those institutions in which all branches of veterinary education are given in a very limited number of university chairs. Such a system is absolutely insufficient.

It is very desirable that in every country the veterinary schools should be state institutions.

Proposition 7 was altered by a proposition of Quivogne to omit the second portion of paragraph 1, which effectually suppressed paragraph 3: and one by Wirz to drop paragraphs 4 and 5 as affected by particular local conditions and unsuited for a general decision. As adopted it reads:

7. The professors of veterinary schools ought to possess diplomas of veterinary medicine.

It is very desirable that veterinarians before being called to the professorship should have practiced veterinary medicine for some years.

REMARKS.

The first thing that strikes one in connection with this subject is the contrast between the Old World and the New. Europe seeks to protect her animal wealth by the creation and maintenance of thirty-three state veterinary colleges. The United States, with practically the same area, and with a wealth in live stock which is fast making her the meat market of the world, has not a single institution of the kind supported and controlled by State or Federal Government. Europe has learned, by a sad experience with animal plagues, that her only safety consists in the creation of educated veterinarians by maintaining a sufficient number of thoroughly efficient establishments, the diplomas of which shall be sufficient guarantee of the knowledge and ability requisite to carry on an effective veterinary sanitary service to care for the cavalry and artillery horses, and to provide everywhere the men wanted for the treatment of her flocks and herds. In the United States, in the absence of any Government college, the public demand for veterinarians has led to the establishment of schools as private enterprises, some of which, like the earlier schools of Boston and Philadelphia, have prostituted their charters by making it a mere expedient for the sale of diplomas to all who would pay the price, irrespective of education or fitness, while others have filled their chairs with men who were themselves destitute of a veterinary diploma, and made them veterinarians by bestowing the diploma of their own institution. The result is that the country swarms with empirics, and that even the possession of a diploma is no guarantee of education or ability. If suddenly called upon to stem a great wave of infection among animals it would be no easy matter for this country to speedily provide the necessary men who could be relied upon for the work. If, again, it were necessary to secure the public health by the suppression in animals of plagues communicable to man, such as anthrax, tuberculosis, glanders, and farcy, milk-sickness, aphthous fever, diphtheria, trichinosis, &c., we have no State accredited school from which we could draw the requisite experts. Physicians are not instructed in the diagnosis and management of these affections in animals, and what have we done to secure reliable veterinarians? The numbers of our horses and cattle are two-fifths those of Europe—the British Isles included—and the number of our sheep and swine is over one-third of those of Europe, including the same islands.

Our latest census makes the value of our live stock in quadrupeds $1,500,000,000, which is, doubtless, like all official valuations, considerably below the mark. This great moneyed interest, liable to injury by plagues, which tend to increase in geometrical progression, is left without that protection which should have its foundation in a national or State guarantee of veterinary education. Such a guarantee cannot be secured by granting charters. These have too often been made the mere occasion of the prostitution of the science to mammon-worship. To furnish it the institution must be placed above the temptation to acquire, and indeed beyond the possibility of acquiring, means by sacrificing the profession. This may be secured by making the veterinary college part of a well-endowed university, and subject to the laws of the same, or it may be made an independent national or State veterinary school, like most of the schools of Europe, under such laws as will preclude the entrance of the debasing influence referred to.

In view of the foregoing recommendations of the international congress, it would be superfluous to enter into the organization of veterinary schools and their curriculum. It may, however, be well to give some further data as to the facilities furnished in the European veterinary schools. It may be permitted me also to hint that we in America cannot abate one jot of the provisions made for this education in Europe, but rather increase them. In sixteen years since the congress at Zurich it has been found necessary in Europe to demand an increase of the period of study by one-third, because of the increasing extent of the fields to be studied. In addition to all this we are to-day confronted by the great question of the life-history of disease germs, which opens up a new world in pathology, and which can be nowhere so appropriately investigated as in a veterinary college. This the Government owes at once to the great live-stock interests of the nation, and to the cause of sanitary science as applied to the human population. The maladies transmissible between man and animals must be investigated through the latter, and from this man will profit directly by the restriction or extinction of these affections, and, indirectly, by analogies with the newly discovered truths in the case of other affections peculiar to the human race.

In five of the state veterinary colleges of the Continent which I have visited the grounds cover a large area, though situated in a city, as at Berlin, Utrecht, Brussels, and Lyons, and are provided with dwellings and offices for the faculty, library, boarding accommodations for students, museums, dissecting-rooms, rooms for autopsies, laboratories for physics, chemistry, pathological anatomy, microscopy, and biology, pharmacy, lecture-rooms and instrument and retiring-rooms for the different departments, surgical operating theaters, furnace for burning infecting products, horseshoeing forge, halls for clinics (averaging 350 by 30 feet each), provided with forge, means of fastening for operations, &c., and separate buildings for the accommodation of the different kinds of hospital patients (solipeds, cattle, sheep, and swine and dogs), and with special stables for those of each kind suffering from contagious diseases. These last were paved with granite or hard-burned bricks, set in cement, and lined for 8 feet from the floor with enameled tile, set in cement, while all the fittings (stall, rack, manger, &c.), were of iron to facilitate disinfection. Then each school had its botanical garden, and in some the different field crops were cultivated, and several specimens of each of the best breeds of domestic animals of the same or adjacent countries were kept for purposes of instruction.

These state veterinary schools further have bursaries for poor but deserving students, the French Government providing no less than 240 of these under conditions which demand excellence alike in deportment and study. The minister of war can further send a certain number of students (in France 60) to be educated free for service in the cavalry and artillery.—J. L.

4. THE RIGHT OF VETERINARIANS TO FURNISH MEDICINES FOR THEIR PATIENTS.

This subject, introduced by Rossignol, apropos of a recommendation of a commission of the French legislature to abolish this right, was shortly discussed, and after securing a statement from the attendant representation of each country in Europe and America, as to the practice in that country, the congress decided as follows:

1. Considering that veterinarians are initiated by the technical studies pursued in their schools into the posology which pertains to the administration of medicines destined to the different species of animals;
2. Considering further that from this point of view they possess better guarantees than do druggists against the dangers which result from erroneous prescriptions;
3. Considering that the right to prepare and sell medicines, especially destined to the treatment of diseased animals, is indispensable for veterinarians who have ready all the medicines necessary for the treatment of the animals to which they are called, and who can furnish the same to their employers at a low price;
4. Considering that a law prohibitive of this is not only useless but opposed to sound economy—

The international congress expresses its opinion that in all countries veterinarians should have the right to prepare and sell medicines destined to the treatment of diseased animals, at least within the limits of their practice, and that it should be forbidden to empirics to keep pharmaceutical substances.

5. TUBERCULOSIS IN ANIMALS.

This subject was most extensively and ably treated by Lydtin, of Carlsruhe, reporter of the commission appointed to bring it before the congress. Unfortunately so much time had already been consumed on the preceding subjects that the congress could not give it the full and deliberate consideration which its overwhelming importance demands. Its full consideration may therefore be held to be deferred until the next veterinary congress shall meet in Paris, when the increased knowledge of the disease will doubtless strengthen views which may to-day be looked upon as in some respects premature. Meanwhile a summary of Lydtin's excellent report, and the action of the congress on it, cannot fail to have a high value. It concludes by proposing for the adoption of congress the following resolutions:

1. Tuberculosis is transmissible hereditarily.
2. It is contagious.
3. It should be included among affections which should be opposed by measures of sanitary police.
4. The measures that ought to be adopted for this purpose are the following:

(*a*.) Every owner of domestic animals must report promptly to the authority charged with this police service every case of tuberculosis, and any symptom causing suspicion of the existence of this affection; he must keep every animal attacked or suspected out of any place where it may be able to transmit the malady.

The same obligation should be incumbent on the steward, or representative of the proprietor, on the person conducting a herd or flock in transit, also on the proprietor of a stable, yard, pasturage, or park where animals are temporarily received.

This report is equally obligatory on veterinarians, and on any person who practices by profession the art of veterinary medicine, on meat inspectors, and upon every person engaged in the trade, of the destruction, utilization, or manipulations of cadavers or their products, if before all intervention of the police he discovers the existence of tuberculosis, or recognizes symptoms which lead him to suspect the presence of this malady.

(*b.*) The appearance of the affection and the particular herd infected ought to be published.

(*c.*) The suspected as well as the diseased animals should be sequestrated, and their slaughter ordered by the police, animals suspected of being infected should be kept in quarantine, unless there are comparatively few, in which case they should be slaughtered by official order. If a large number are suspected, they may be fattened and sent to the abattoir as quickly as possible.

(*d.*) Infected stables and other places should be under the special surveillance of the police for an entire year, counting from the last case of the disease. The sale of beasts suspected of infection should be interdicted, unless it is for slaughter, and under the inspection of a veterinarian.

(*e.*) The place occupied by a tuberculous animal ought to be cleansed and disinfected, the animal having been previously removed; it ought to be the same when the malady has disappeared from stables and other closed places in which tuberculous animals have been kept; it is only after disinfection that the prescribed police measures should be removed. During the whole course of the panzootic the stables should be especially well ventilated.

(*f.*) The flesh and viscera of a tuberculous animal can only be utilized for consumption when the disease is found *in the cadaver* in its incipient stage, when the lesions are confined to a very small portion of the body, when the lymphatic glands are still free from all morbid tuberculous lesion, when the tuberculous formations have not yet undergone softening, when the flesh presents the characters of meat of the first quality, and when the animal is in a good state of nutrition at the time of slaughter.

It should not be permitted to remove the flesh of tuberculous animals, admitted to consumption, out of the locality where they have been slaughtered, and it should not be offered for sale in the ordinary butcher's stall.

Every quarter of meat and all viscera showing lesions of tuberculosis, as well as the flesh of any other animal in which there is found at the necropsy a tuberculous infection more pronounced than that referred to above, should be watered with petroleum oil, and afterward buried under police supervision. The extraction of fat by cooking and the utilization of the skin may be permitted.

The inspection of every animal attacked by tuberculosis should be made by a veterinarian, who alone should decide if the flesh is fit for human consumption.

(*g.*) The milk of animals suffering from tuberculosis, or suspected of it, should not be consumed by man nor certain animals. The sale of such milk should be severely interdicted. The milk of animals suspected of infection should only be used after boiling.

(*h.*) Under proper safeguards against its abuse, it would be proper to furnish indemnities for cattle slaughtered by official order in consequence of tuberculosis, also for those that have died from this malady, and for those found to be tuberculous after they have been killed for human food. The indemnity may be paid out of the state treasury, or to create the requisite funds resort may be had to an obligotary insurance.

(*i.*) Violations of orders relative to measures preventive and repressive of tuberculosis should be punished.

(*j.*) As a safeguard of the public health against the dangers which threaten it through the consumption of flesh furnished from diseased animals, of stale or putrid meat, and of falsified sausage and mince-meat there should be established in every commune a competent service for the inspection of meats.

(*k.*) Establishments which make a specialty of furnishing milk for invalids, or for infants, ought, as regards these milch animals, to be submitted to a constant control confided to veterinarians officially designated for this purpose.

In recommending these resolutions for the adoption of congress it has been felt that they are fully called for in view of the danger which has long threatened the interests of stock owners and the health of the community, and which constitutes a veritable calamity.

Lydtin begins by tracing the history of tuberculosis, showing how like other diseases, and notably glanders, it appears in forms that are not recognized as identical with the common types, and how, in consequence, its true characters, its prevalence, and, above all, its contagious property were ignored, until anatomo-pathological observations showed the identity of the lesions in different organs, and biological experiments established the true nature of the disease germ.

He thinks Moses refers to phthisis in the word *dürre*—(leanness) Lev. xxii, 22—and Columella, as phthisis (De re Rustica); the same idea is embraced in most of its common designations—as pining, consumption-decline, *Schwinden*, *Schwindsucht*, phthisis, *ulceration pulmonaire*, *Lungen verschwärung*, *lungensucht*, *lungenfäule* sufficiently illustrate.

Another form has been named and classified, on account of sexual aberrations, *nymphomania*, *satyriasis*, *Stiersucht*, *geilesucht*, *Monatsreiterei* (German), *ninfomania*, *furore uterino* (Italian), Brummel (Swiss), bulling (English).

Another form has been named, from the more or less firm excrescences which appear on the serous membranes and skin—*Perlsucht*, *Hirsesucht*, *Meerlinsigkeit*, *Zäpfigkeit*, *Kranichtkeit*, *Rindshammen*, *Traubenkrankheit*, pearl disease, knots, kernels, grapes, angleberries, clyers.

The supposed syphilitic character of the disease produced the following names: *Franzosenkrankheit*, *Francausowitost pri Krawach*, *Lustseuche*, *Unreinigkeit*, *venerie et morbus gallicus boum*.

The implication of the glands and the sarcomatous and fibrous character of the growth has given rise to still other names: *Drüsenkrankheit*, *malattia glandulare*, *sarkomdyskrasie*, *cahexia boum sarcomatosa*, *sarcomatosis infectiosa*, *sarco-tuberculosis*, *tuberculosis fibromatosa*, *rindstuberculose*, &c. Again, as it appears in the pig in the form of scrofula, it has been called scrofula tubercle.

No wonder that the identity of all those forms of the disease was slow to be recognized, and that pathological anatomy and inoculative experiments had to be invoked to determine it. The name to be preferred is the generic one *tuberculosis* (or *tuberculosis panzootica contagiosa*), and yet this must not be held to imply that the nodosity (tubercle) is a constant and pathognomonic feature of the disease.

The manifestations of the disease are entered on fully, showing that after the preliminary slight fever (marked symptoms often subside and of the local lesions are confined to certain non-vital organs) there may be comparatively little sign of illness for months or years. The flow of milk may be abundant, and though variable yet of no fixed quality, and the animal may breed, work, or even fatten without suspicion. As the diseased processes extend over the system febrile symptoms reappear and tend to assume a remittent character, the temperature becoming abnormally low in the morning and high toward night, and at the same time wasting advances more or less rapidly. Enlargment of the cervical lymphatic glands, irregular appetite, tympanies, colics, constipations, and diarrheas, and indications of lesions of the respiratory organs, are especially common. In certain cases there may be muscular or nervous disorder, cramps, paralysis, wryneck, epilepsy, turning in a circle, coma, blindness, in others disease of the testicles, or udder, of the bones, joints, and skin.

The affection may prove fatal in less than three months, or it may last for an ordinary lifetime. It may induce other diseases of the organs in which it is located, and thus greatly complicate the symptoms or hasten a fatal result.

It follows that the disease is not always easy to diagnose. Yet its symptomatology is quite as advanced as that of many other diseases, and with the recent demonstration of its germ—*bacillus tuberculosis*—it is sufficient for the purposes of veterinary sanitary police.

The pathological anatomy of tuberculosis is more perfect. The lesions most constantly met in the cadaver are the neoplasms on the pleuræ and peritoneum. These vary in size from a millet seed to a pea; they are single or united in bunches (grapes), pedunculated, polypoid, or warty, red, flesh-colored or brownish-yellow, and of most varied consistency. Very often the center of the soft nodosity is deep red, while that of the hard one is yellowish and caseated, or of the consistency of mortar. According to Virchow they appear first as little nodosities or pearls in groups projecting slightly from the surface of the serous membranes; later they become pedunculated, remaining connected by vascular bands of connective tissue; still later earthy salts are deposited in them, and finally they soften, undergoing fatty degeneration, and become like a thick mortar.

Changes in the lymphatic glands of the head, neck, chest, abdomen, &c., are also present in all but the most exceptional cases. These are swollen, and of a dull, yellowish color, impregnated with juice or pigmented. They show hæmorrhages as large as a pin's head, irregular enlargements, and indurations. On section the surface shows numerous infiltrated points of the size of a millet seed to a pea, of a grayish yellow or whitish color, and the consistency of cheese or mortar. Larger centers of irregular shape, but the same characters, are also met with.

The lungs in most cases present similar lesions in nodules and nodosities in all stages from the simple hemorrhagic point to the caseous or calcareous mass, also connective tissue neoplasm, which obliterate the pulmonary lobules and attain considerable size, and finally caseous masses in the midst of lung tissue, otherwise unaltered. Sometimes the nodosities of the pleura covering the ribs adhere to those on the lungs, and they may become continuous into the lung tissue for an indefinite distance.

The softened nodosities may open into the pleural sac with fatal effect, or into the bronchia, causing a grumous discharge from the nose and mouth, and when empty they form cavities—vomicæ.

The nodules are common on the mucous membranes of the trachea, larynx, pharynx, and gullet, and in the submucous tissue of these parts, and softening and discharging they form funnel-shaped ulcers, which become confluent and cause deep and extensive sores invading the subjacent cartilage and other tissues.

Sometimes the brain and spinal cord are invaded, but especially the pia mater and arachnoid, giving rise to the most varied nervous symptoms, and passing through the same changes with an especial tendency to puriform softening when in the brain substance.

Tubercular deposits in the coats of the bowels are found in cattle in the form of nodules varying in size from a pin's head to a hempseed on the inner surface of the peritoneum (Niklas), and in pigs in the forms forms known as scrofula or caseous enterites.

The miliary nodules and aggregations of them are also found in the liver and spleen, less frequently in kidneys and bladder, and in the generative organs (tunica vaginalis, cord, testicle, uterus, ovaries, Fallopian tubes, vagina), and in the mammary glands. The muscles are occasionally the seat of tubercle, and the bones rather frequently so. The neoplasm takes place by preference in the cancellated tissue of the extremities of long bones, and in that of the bones of the cranium, and the spines of the dorsal vertebræ.

The relative frequency of the more common seats may be deduced from the following table of 1,596 cases observed in Baden:

	Per cent.
Lesions of the lungs only	21
Lesions of peritoneum and pleura only	28
Lesions pulmonary and pleural	39
Lesions of generalized tuberculosis	6
Lesions of generative organs only	3

Microscopically the tubercular products are composed of—

(*a*.) Excessive growth of new connective tissue which, in the lungs, may amount to 55 to 100 pounds.

(*b*.) Connective tissue growths interspersed with centers of degeneration: *first*, hemorrhagic points; *second*, small vitreous looking masses, and, *third*, soft caseous collections. Both these forms show a strong propensity to calcification, and in bones to genuine ossification.

(*c*.) Sarcomatous neoplasms or tubercular nodosities. These have a vascular stroma of connective tissue inclosing masses of round lymphoid and fusiform cells. The peripheral cells have a clear outline, and there are few free nuclei, while in the center the cells become opaque and indistinct with little protoplasm and shriveled nuclei and free nuclei and granules abound. These also are often calcareous.

(*d*.) Tubercles varying in size from a millet seed to a hen's egg. These appear in the parenchymatous tissue of the lung, &c., while those formerly described affect rather the surface and the serous membranes. These are at first small and translucent, but increase by juxtaposition and confluence, and being exclusively cellular become early calcified, or more frequently caseated. These are much more numerous in an infected tissue than any product likely to be mistaken for them.

(*e*.) Ulcers of various forms and dimensions. In all these lesions there is the same initial change—proliferation of the connective tissue. The variations in the nature of the lesions depend on the different activity of the intercellular substance and the connective tissue and endothelial cells, on the disaggregation of the elements and the occurrence of ulceration. Thus the fibrous growth is usually greatest where connective tissue is most abundant, as in bones, cartilages, and interlobular lung tissues, while the more cellular and rapidly disintegrating growth occurs in connection with epithelial structures, as in the air cells and on the inner coat of blood-vessels. This tendency to attack the connective tissue and lymphatics, and to show products varying according to the nature of the surrounding structures is common to other infectious diseases, and notably glanders, chronic lung plague, actinomykosis, &c. As in these cases, too, the morbid process is first localised and only becomes diffused when it has attained a certain local intensity. Again, it agrees with other infectious diseases in being favored by certain unhygienic conditions, as damp pastures, close filthy buildings, overcrowding, poor food, excessive work or milking, &c., yet is not absolutely dependent on any one or more of these, nor due to these alone. As in these other affections there is the disease germ—*bacillus tuberculosis*—the presence of which is essential to the development of the disease, and its recognition completes the diagnosis.

Again, there is a special constitutional predisposition in animals having an excess of connective tissue and of lymphatic development as in cattle and swine.

Climate seems to have much effect, as the disease is virtually unknown in northern and arctic climates—Iceland, Northern Norway, and Sweden, Finland and Lapland—and very common in the temperate and tropical regions.

(While frost doubtless chains up this germ when out of the body, as it does others, it must not be forgotten that the paucity of cattle in the extreme north will even more tend to retard the propagation of this disease. Other countries formerly free from tuberculosis have now, by the influx of consumptive patients, and, in some instances, by the greater density of the population, become extensively affected with this disease, as witness the Hebrides, Australia, and our northwestern States and Territories.—J. L.)

To the same effect speaks the great prevalence of tuberculosis in dairies near cities where the stock is often changed and new stock is being constantly purchased, and its almost complete absence from districts exclusively devoted to breeding and never importing strange stock. This is but a repetition of what is known of other contagious diseases. Wild races, too, living in the open air are largely exempt.

Heredity as a cause of tuberculosis.—In favor of the heredity of tuberculosis Lydtin quotes from over a score of veterinary authors. The congenital presence of the disease is proved by quotations from König, Stirnimann, Adam, Butscher, Virchow, Semmer, Jessen, Fischer, Müller, Zipplius, &c. The infrequent recognition of the disease in veal calves is noteworthy, being generally only a fraction of 1 per cent., but it must be added that tuberculosis in the dam, affecting the fetus, usually determines the death of the latter, followed by abortion, and that of those in which the disease stops short of this the tubercles often rest circumscribed and inactive in an unimportant organ until the young animal is more fully developed, or even grown up.

(I have repeatedly seen abortions as the first indication of tuberculosis in a herd, and calves of healthy breeds infected by milk grow to maturity and then fall victims, where the ancient calcified products were found side by side with the recent. It is further to be noted that the calves more severely affected perish of indigestions, diarrheas, &c., and are buried by the owners without any notification of the authorities. These, therefore, cannot be fattened for veal.—J. L.)

Göring noticed that in bovine tuberculosis 123 were infected by the dam and 43 by the sire. The hereditary cases constituted 12 per cent. of all cases of the disease. All veterinary and agricultural writers attribute a most disastrous influence to *in-and-in breeding* (in infected families).

The special predisposition of animals with much loose connective tissue has been already referred to. It is further noticeable that this is a prerequisite to aptitude to fatten, and it is notorious that many families of our best breeds of meat-producing animals are affected with tuberculosis, while the disease is less prevalent among unimproved races.

(In this connection we must not ignore the *close breeding* of the latter, their residence in a hot forcing atmosphere, and their preservation for breeding purposes, even when manifestly unhealthy, nor the free open-air life and the prompt disposal of unthrifty beasts among the less valuable breeds. The disease being due to a specific germ, it should be limited by the repression of that and not by abolishing in our meat-producing animals those qualities which constitute their value.—J. L.)

The conclusions as regards heredity are thus:

1. That heredity is not without influence upon the propagation of tuberculosis.
2. That this malady is transmitted alike by the sire and the dam.
3. That the transmission of the morbific principle to the ovule or fetus in course of development, is a cause of sterility in the parents, and frequently occasions abortions and premature parturitions.
4. That a fetus affected with tuberculosis rarely attains complete maturity or comes into the world in conditions of normal viability.
5. That notwithstanding these facts, we cannot deny the possible birth (perhaps in great numbers) of tuberculous descendants which can develop and multiply equal to animals, in perfect health and without any predisposition.

Contagion as a cause of tuberculosis.—Lydtin refers to the above proof of the transmission of the disease through the ovum, sperm, uterine secretions, &c., and proceeds to offer further evidence of direct contagion. He quotes Ruhling, Krunitz, Fromage, Huzard, Spinola, Crugel, Lafosse, Villemin, Dupont, and Zaugger, in support of this, and advances the following proposition:

> The morbific principle can enter the system either by the respiratory or digestive apparatus. The inspired air and the forages and drinks can act as vehicles; it may also be transplanted by way of the generative organs (coitus), or by wounds, accidental or experimental.

Instances of infection by cohabitation are quoted from Stahl (to 4 stud bulls by a newly-bought tuberculous one of different blood); Renner (to cow and calf from tuberculous cow of different blood); Fischer (heifer from tuberculous cow and 3 cows and ox from tuberculous cow of alien blood); and Jaum (11 cows from a new tuberculous one); Ross (4 cows from a new tuberculous one); and others by Viseu, Zundel, Grad, Haushatter, Leutz, Huzard, Tessier, D'Arboval, Remy, and Hugues. Cases of infection through *food* are quoted from Jessen (calves sucking sick cows, died in six to twelve months); Volkers, Lehnert (2 pigs of healthy parents in one month after feeding unboiled milk of sick cows); Bromley, Walley, Fleming, Zippilius (calf sucking tuberculous dam died of diarrhea with circular, belt-like ulcers of small intestine); Gerlach (many calves and pigs infected by milk of sick cows), &c.

Transmission by *coition* is less definite, but is inferred from the occurrence of tuberculosis in the womb, Fallopian tubes, and ovary of the female (Adam), and the testicles of the male (Schlotterer), also from the abortions in tuberculous herds.

Of *transmission by raw surfaces*, all the cases of experimental inoculation are instances. Lydtin took lymph from lung affected with tubercle and lung plague, but carefully avoided any point where tubercle could be detected, and avoided also the blood as far as possible. With this he inoculated 10 cattle, 5 of which, when killed twenty-three days later, showed numerous distinct miliary tubercles in the inoculative swelling, and one tubercle in the right lung; the remaining four showed tubercles of older date in the lungs.

Toussaint inoculated a tuberculous cow with cow-pox, furnished by a healthy heifer, and eight days later from the resulting cow-pox vesicles inoculated four rabbits and a pig. All of the rabbits became tuberculous in two months.

In the field of the experimental transmission of tubercle, the work has now been extensive and the results most convincing. Villemin, Gerlach, Chauveau, Colin, Soujou and Court Paul, Günther and Harms, Rivolta and Peroncito, Bagge, Bollinger, Kohne, Semmer, Biffi and Verga, Bouley, Peuch, Aufricht, Toussaint, and others have contributed in varying degrees to the solution of the question, and the grand result attests indubitably the communicability of the disease.

Günther and Harms conveyed the disease to 5 rabbits by making them *breathe* the expired air from tuberculous cows. Tappeiner conveyed it to dogs by diffusing the tuberculous sputa of man in spray in the air they breathed.

Experiments in feeding the infecting matter are tabulated as follows by Johne.

Animals experimented on.	Number of animals.	Results.		
		Affirmative.	Negative.	Doubtful.
		Per cent.		
Horses	1	0.0	100.0 *per cent.*	0.0 *per cent.*
Calves	5	100.0	0.0	0.0
Sheep	35	51.4	12.9	3.7
Goats	13	84.6	15.4	0.0
Swine	60	65.0	18.3	16.6
Rabbits	171	31.2	66.5	2.3
Dogs	20	25.0	75.0	0.0
Cats	9	55.5	44.4	0.0
Guinea pigs	6	83.3	16.6	0.0
Pigeons	2	0.0	100.0	0.0
	322	43.5	51.1	5.0

Table giving the results in the same animals according to the food eaten.

Material fed.	Results.		
	Affirmative.	Negative.	Doubtful.
	Per cent.	*Per cent.*	*Per cent.*
117 tubercular matter from heifer	61.5	34.2	4.3
46 raw flesh of tuberculous cows	13.1	86.9	0.0
91 milk of tuberculous cows	30.7	59.3	1.0
1 milk of tuberculous rabbit	100.0	0.0	0.0
25 tubercular matter of man	36.0	64.0	0.0
33 tubercular matter of pig	53.0	47.0	0.0
2 tubercular matter of sheep	100.0	0.0	0.0
2 tubercular matter of rabbit	50.0	50.0	0.0
3 tubercular matter of ape	100.0	0.0	0.0
5 tubercular matter of birds	100.0	0.0	0.0

Johne concludes—

1. That tuberculosis can be transmitted from animal to animal and from man to animal by feeding on tuberculous substances, but this mode of transmission is much less certain than by inoculation.
2. The materials which most certainly transmit tuberculosis by gastro-intestinal ingestion are tuberculous matter taken from the lungs, pleuræ, and lymphatic glands; milk of tuberculous animals, as regards its contagious properties, must be placed near to these. Infection by tuberculous matter taken from man is less certain than by that taken from animals.
3. Infection is less certain from the ingestion of muscle than by the substances indicated under 2, and yet it occurred in seventy-six cases in the above named experiments.
4. Calves, sheep, goats, and swine present the greatest susceptibility to tubercular contagion; the pretended immunity of carnivora is not so pronounced as certain authors have alleged.

Gerlach found that of 46 animals fed raw tuberculous matter 35 became infected; that of 35 fed raw muscle from tuberculous subjects 8 became infected, and that of 15 fed cooked tubercular matter 10 were infected.

Bollinger produced tuberculosis in pigs by prolonged feeding of the milk of tuberculous cows.

The subcutaneous inoculations of Villemin and his successors were hotly disputed on the ground that they gave rise to lesions analogous to those produced by inocculation with non-tubercular matter. These were practically settled by the intraocular injection of white rabbits with tubercular matter by Cohnheim, Salomonsen, Hansell, Deutschmann, and Baumgarten. After an incubation of twenty to thirty days there appeared in the pigmentless eye distinct tubercular nodules, and this was followed by a generalized tuberculosis. Baumgarten developed tuberculosis in the eye by injecting the blood of tuberculous animal infected by inoculation. In test experiments with the blood of healthy animals the eyes remained sound; when he used the blood of septicæmic patients intense inflammation of the eye ensued, but never tuberculosis.

Toussaint found the tubercular lung products of cows constantly infecting to rabbits and pigs after they had been subjected to 55° to 58° C. in a water bath, and even after they had been roasted like a beefsteak in the gas flame. He found the nasal discharges, the saliva, and the urine infecting, and as already noticed the lymph of a vaccine vesicle. Lydtin concludes:

> That tuberculosis is contagious, like glanders or lung plague, and that contagion fills a more important rôle than heredity in the propagation of the disease.

As showing the identity of tuberculosis in man and animals, Koch's demonstration of the bacillus tuberculosis must occupy a prominent place. The disease had already been proved a hereditary and an infectious one, and this organism, found in the growing tubercle of man and animal alike, suggests itself at once as the morbific germ. It is found alone and unmixed with any micrococcus, in deep seated tubercles, which have had no exposure to the air, while in sputa, vomicæ, and other tubercular products exposed to the air a multiplicity of other organisms abound. In all cases of rapidly growing tubercles the bacillus is present in great numbers, while in those of slow formation they are scanty. These bacilli have a length of half the diameter of a red blood globule and a breadth of one-fifth of their length; they are motionless and form spores within the body even during the life of the animal.

After many attempts Koch succeeded in procuring a pure culture in blood serum of cow or sheep in a preparation of gelatine, on which the bacillus appears as fine scales at the end of two weeks. They grow so slowly that it is only at the end of the third or fourth week that the mass attains the size of a poppy seed. It does not develop save at a temperature of 30° to 41° C. These peculiarities of culture identify the parasite.

The bacillus, whether derived from the tubercle of man or that of animals, always shows the same form and the same habits during culture, and on inoculation has produced the same pathological lesions, implying the essential identity of the two.

By numerous carefully controlled experiments Koch has proved that it is impossible to produce the typical alterations of miliary tuberculosis by the inoculation of other matters than the bacilli; in these experiments he has taken all necessary precautions to avoid confusion with spontaneous tuberculosis, and to exclude all infection from any accidental source of the subjects on which he operated. He concludes that the presence of the bacillus in the tuberculous masses is not a simple concomitant of the tuberculous process, but the cause, and that we must recognize in the bacilli the cause of tuberculosis, hitherto unsuspected, but now evident in the form of a vegetable parasite.

Koch has found this parasite in all forms of scrofula and tubercle in man and animals, and in 109 inoculated subjects (rabbits, guinea pigs, and cats) in the nodosities of the lungs.

Add to this that Villemin and Klebs have demonstrated that the tubercle of man, on inoculation, produces phthisis pulmonalis in animals, and that this inoculated phthisis is transmissible by inoculation to other animals.

Johne mentions a case of successful inoculation of tubercle from man to man, and Staug a case of the accidental infection of the son of healthy parents by habitual drinking of the warm milk of a tuberculous cow.

Another argument in favor of the identity of the disease in man and animals is the perfect analogy of the disease as regards heredity and contagion in the two.

The heredity in man is shown by the presence of the disease in the fetal offspring of tuberculous parents. Walshe records the frequency of abortion and sterility in tuberculous patients. The doctrine of the contagion of tuberculosis in man has been sustained by Galen, Norton, Swieten, Home, Maret, and many later observers. Instances are quoted of infection through clothes and beds, and from husband to wife. Wichmann, in 1780, said that one death in six in the population of Zurich was from tuberculosis, and details the different channels of direct and indirect contagion, going so far as to advocate a supervision of the sale of old bedding and clothing. Cullen, at the same date, speaks of its propagating itself most readily in the warm climates of Southern Europe, where (Italy, Portugal) to the present day the clothing, bedding, and other agents used about a person deceased of phthisis are invariably destroyed. Lydtin concludes:

1. That tuberculosis has been observed in all warm-blooded animals submitted to domestication or deprived of their liberty.
2. Tuberculosis of animals and of man present analogous manifestations in the living and in the cadaver.
3. The course and termination of the two maladies are the same in man and animals.
4. The tubercular masses, and, above all, the expectoration of phthisical men determines tuberculosis in animals when these masses are introduced into the latter by the respiratory or digestive apparatus, or by a deep wound. Tuberculosis inoculated from man to animals can be thenceforward transmitted from one animal to another, producing in all cases tuberculosis.
5. Tuberculosis in man and in animals is transmitted by heredity.
6. Tuberculosis is contagious to man as it is to animals.
7. There are clinical observations proving the transmission of tuberculosis from animals to man through the use of the milk of phthisical animals.
8. Tuberculosis of man and that of animals are rare in the cold climates, and even appear not to be developed. They are more frequent in warm climates; the geographical distribution of the two maladies is almost the same.
9. It is demonstrated that a pathogenic microbe having the same morphological and biological characters, exists in the tubercle of man and in that of animals. This organism, whether developed in man or animals, can produce tuberculosis when, cultivated in a state of purity, it is transmitted to a susceptible animal.

It is only necessary to add that tuberculosis in animals tends to concentration in the large dairies and feeding establishments which supply the great centers of population. The farmer, watching closely the animals he has owned since their birth, is led, by the instinct of self protection, to sell off those that show symptoms of failing, and these usually go to the large establishments near the cities, there to be crowded in close buildings with many others, to which they in turn convey the infection. If in a dairy, these supply milk for the population at large, including the susceptible infants and invalids, and finally all or nearly all of such animals find their way to the butcher's stall, when they can no longer be utilized for other purposes.

(To show that America is no better than Europe in this respect, it may be stated that 29 per cent. of the adult males dying in New York City are tuberculous, and that in certain of the herds that supply that city with milk, 20, 30, and even 50 per cent. are affected with the same disease. In some country districts of New York can be shown large herds with 90 per cent. the subjects of tuberculosis. Were all the known facts published concerning the ratio of tuberculosis in certain communities and in the herds supplying their meat and milk, there would be a testimony far more telling than even the striking example of New York City. One stands appalled at the immensity of this evil, covering as it does the entire country, threatening at every step the health of the community, and crying loudly for redress.)—J. L.

Nothing has as yet been said as to the propagation of tuberculosis from man to animals. In the large stables adjoining cities the feeding of products from the kitchens, mixed with excretions of man, doubtless contributes to this, and the contrast of the frequency of tuberculosis among these and its absence among the wild oxen of Hungary suggests the importance of such a factor.

PREVENTIVE MEASURES CALCULATED TO CHECK THE EVIL RESULTING FROM THE CONSUMPTION OF THE MILK AND FLESH OF TUBERCULOUS ANIMALS.

Lydtin refers to the law of Moses, under which the pining (dürre Schwinden) animal could not be eaten; to the Mischna, which expressly condemns carcasses the lungs of which cannot be inflated, or have attached growths; to the continuation of this law among Christians until the third century; to the Roman masters of markets (ædiles), who saw that bad and corrupt meat was thrown into the Tiber; to the church law published in the tenth century against the consumption of diseased meat; to the ancient laws of Italy, France, Spain, and Germany against the sale of diseased meat, and to the more modern statutes on the same subject. He quotes from Zückert (1775) the case of the death of twelve students from eating the flesh of cows of which the viscera were covered with a great number of vesicles, tubercular nodules, and purulent tumors. In furnishing such meat for the soldiers a French butcher was, in 1716, condemned to nine years' exile, a fine of £5,000, and permanent prohibition from engaging again in the same trade.

A German law of 1732 imposed a penalty of 50 rix thalers, with the addition, in certain cases, of corporal punishment for the sale of such meat or for evading its inspection. About 1764 the doctrine of the identity of tuberculosis and syphilis was contested by Zink, Ruhling, Heim, Graumann, and Zwierlein, and the German law was abrogated while that of Lower Austria was modified so as to condemn only advanced cases. In Southern Germany, Austria, and Switzerland the meat of tuberculous animals has always been more or less forbidden. In the main, however, it is only advanced cases, those with profound and extensive lesions, that have been condemned. The same has been the case for France and Belgium. France was the first, in 1810, to abolish private slaughter-houses in large and medium sized cities, and to introduce that essential condition of all effective inspection—municipal abattoirs. Later this has been adopted by nearly all the cities of Europe and a thorough inspection rendered possible.

Cooking of diseased meat in general was held by Payen, Renault, and others to render it innocuous. Its value as regards tubercular products was first investigated by Gerlach, who also led in the inquiry as to the danger from the different portions of a tubercular system. He found that the morbific matter resided mainly in the tubercle, from which it spread slowly along the lymphatics to the next lymphatic gland, and then still along the same vessels to more distant glands, and finally it became generally distributed. He further attached much importance to the multiplicity of caseous centers, especially in the lungs, as the breaking down of the nodosities as well as the extension and increase in numbers of the tubercles imply a greater danger of the contamination of the flesh. Emaciation is another indication of the general action of the poison. A very advanced condition of any one of these morbid states should forbid the use of the flesh as food.

The Veterinary Council of Germany (1875) and the Berlin Veterinary School (1878) respectively pronounced on the subject with great reserve, virtually holding the matter still *sub judice.* Johne held that the mere extension of tuberculosis from the first seat to the neighboring lymphatic glands did not imply unwholesomeness in the flesh, and that it was only requisite in such cases to cut out the tubercles, enlarged or diseased glands and adjacent connective tissue. He pronounced the flesh as dangerous when the extended lesions implied an infection of the blood; when, for example, from primary deposits elsewhere there are resulting tubercles in lungs or pleuræ. Zundel, basing his opinion on the failure of Koch to find the bacillus in any part save in the tubercular centers, concludes that no meat should be condemned except that furnished by animals emaciated and thoroughly infected.

That measures protective of the public health should be taken is indisputable. The demands of those who demanded new experiments have now been realized. The experiments demanded have been repeated, and when practiced with all needful precautions have given positive results. The bacillus of tuberculosis has been isolated, cultivated, and transmitted by inoculation, and whatever the soil from which it has been transplanted (man, animals, gelatine preparations, &c.), it has always determined the development of true tuberculosis in the inoculated animals, and this tuberculosis has proved transmissible from one inoculated animal to another. Tuberculous meat is certainly infecting. How, then, can we protect the community against its effects? One says: *by thorough cooking;* another, *by confiscating the diseased meat.*

While the *boiling temperature* is destructive of the tuberculous bacillus we cannot rely on meat being heated to this temperature. In North and Middle Germany people continue to eat raw meat, notwithstanding the frequent outbreaks of tuberculosis. Again, in ordinary cooking the center of the meat often remains below 212° F., the color of the blood indeed frequently persists. The proposal to sell this meat only in separate stalls at a low price, so that the purchasers would be warned to cook it thoroughly, would be ineffectual, as it would not change the general habit of cooking, and above all it would furnish no safeguard against its careless preparation in public institutions and elsewhere where it is used on a large scale.

Confiscation, applied to all cases, would be far more effectual, and with competent inspectors this could be carried out; but in practice it is surrounded by numerous difficulties. Diagnosis is easy, and with a single municipal abattoir all cases should be detected, but it is found that all the infected do not come to such abattoirs. In Baden 20 per cent. of these are killed elsewhere. An attempt to apply the law stringently in Mannheim resulted in an organized effort to thwart the officers of justice. Stock owners refused to sell to the city butchers unless relieved of all responsibility as to the soundness of the animals, and the butchers declined to use the city abattoir and insisted on handling only dead meat which had been killed outside. The opposition even extended to the consumers, on whom the price had been raised, and the vigorous measures were finally abandoned.

If the difficulty is met by inspection of herds a considerable expense will be incurred and other objections invoked. In short, a sound public spirit is essential to the maintenance of any really effective work.

Another difficulty arises from the degree of infection. If all tuberculous carcasses are seized the way is plain, but if a selection must be made disputes will constantly arise over the interminable shades of difference in the various subjects. The delicacy of the question to be pronounced upon in these cases would demand for each commune a veterinarian exclusively devoted to the work of inspection, but this would add considerably to the expense incurred.

Turning to milk, it cannot be denied that boiling would give the required guarantee of safety; but in the *milk-cure* establishments and where this liquid is produced for infants, it is sought fresh and used without boiling, and yet these places seek for the heaviest milkers, and often secure tuberculous cows. For these, and indeed for all dairies, there should be a State control of the milch animals and their products.

In the present state of public opinion it may be impossible to apply measures that will be really effective in preventing the sale and use of tuberculous meat and milk, yet, where already applied, the preventive measures have proved so beneficial that we must advocate their maintenance and extension. It is encouraging to know that public opinion is gaining on this matter, so that ere long more effective measures will be sustained.

The present difficulty resides in the inevitable imperfection of any control of meat and milk by themselves, and that any permanent surveillance of the cattle without the concurrence of the owners will furnish no guarantee commensurate with the gravity of the danger.

The difficulties of the case will be largely met if the State *will deal with tuberculosis in animals as with other contagious diseases.* The owner, well accustomed to the effects of the disease and practiced in weeding out the consumption and sending them to market, can be called upon to report all cases to the authority. The veterinarian can easily diagnose the disease, or in the few uncertain cases can decide positively at the autopsy. He can further, as in the case of glanders, maintain a prolonged surveillance over the inmates of that stable, and above all on those related by proximity or by blood to the animal slaughtered. Thorough disinfection of stables, utensils, clothing of attendants, &c., can be carried out. If the great majority of a herd are infected, the apparently sound may be taken to another building. The diseased and suspected may be slaughtered at once or after a few weeks' fattening. The sale of the milk and flesh of all tuberculous animals should be forbidden. Finally the owner can be indemnified for every beast confiscated. This alone will secure the co-operation of the owner, without which all efforts will be very partial and imperfect.

In view of the insidious progress of the disease it is desirable even to furnish an indemnity for the carcass of an animal found to be diseased after slaughter. This will do more to check the clandestine sale of tuberculous meat than the most rigid administrative control of the abattoir and butcher's stall.

This indemnity will remove all injurious pressure from the veterinary inspector, and enable him to decide with a safe margin what meat and milk can be sent to market without injury to human health.

The indemnity may be a tax on the State, on the commune, or on the stock owners and butchers who will eventually profit most by the suppression of the disease.

ACTION OF THE CONGRESS.

On account of the short time available for the consideration of the question, Wehenkel opposed any action at the present congress, but Quivogne, Larmet, Aune, Rossignol, and Bouley urged a decision of the question, "*In what cases can the meat be used for food?*" This was agreed to.

Lydtin furnished a resume of his report and concluded by presenting paragraph *f* of his resolution for the acceptance of congress.

Bouley held that one of the most difficult questions in practice was what an inspector should do with the carcass of a tuberculous beast. It is established that tuberculosis is a malady dangerous to the animals which are able to contract it, not only by the respiratory passages, but also by ingestion into the stomach and by inoculation in any part of the system. He referred to Toussaint's experiments of which the results were frightful. The tuberculous element did not reside only in the tuberculous lesions, but is diffused through all the tissues. The juice of the flesh of a tuberculous animal, even after it had been heated to 50° or 60° Cent., that is the temperature of roasting beef, has proved virulent for the ox, pig, cat, rabbit, &c., even when given only in a virulent dose, a small dose; large doses are not necessary. Considering the facts in this light we ought to establish no degrees in tuberculosis; when it exists it renders the consumption of the flesh dangerous. He is convinced that tuberculosis, whatever may be its degree, should entail the condemnation of the meat; it can only be utilized if well cooked.

Tuberculosis of man is certainly contagious, as is known to all the world, being principally transmitted from husband to wife. The disease is very widely spread, affecting one-fifth of our population. We must look to butcher meat as one of the principal causes of this great prevalence of tuberculosis; it is probable that the infection enters by the digestive passages as in the experiments of the laboratory. He concluded by proposing the following amendment:

> Tuberculosis being experimentally demonstrated as a malady transmissible by the digestive canal and by inoculation—
>
> The congress declares that meat provided by tuberculous animals should be excluded from the food of man, whatever may be the stage of the tuberculosis, or the apparent qualities of the meat.

In his judgment, stock owners should be indemnified, and butchers urged to establish mutual insurance societies.

Van Hertsen detailed the practice in the Brussels abattoirs, long in use, and which he had set forth in a paper laureated by the Veterinary Society of Eure and Seine in 1869. He seizes all lean tuberculous animals, all having numerous and generalized tubercles, or with tubercles softened, caseous or purulent, lesions which create the suspicion of general infection of the system. When their lesions are grave and multiple, whatever may be the quality of the meat, seizure is always ordered; when the tubercles are localized and recent, and provided the carcass is sufficiently fat to guarantee good meat, it is allowed to be used. As to the danger from unboiled milk he had with Degive, in 1868, presented a paper to the Belgian Academy of Medicine on the frequency of tubercle in the mammæ of cows, but the academy discarded the subject without discussing the important question of hygiene therein presented to them.

Wirz proposed an amendment of *f* in Lydtin's report, that in place of "to a very small part of the body, when the lymphatic glands are still free," &c., read "to but a small part of the thoracic or abdominal viscera, when the lymphatic glands which do not belong to these are free," &c.

Van Hertsen believes that tuberculosis can invade the lymphatic system when the disease is yet in its earliest stage. He has observed that the gland situated between the first and second rib is tuberculous eight times out of ten. This gland is of the greatest importance in determining the quality of the meat, as it establishes the fact of tuberculosis without the necessity of examining the viscera. It serves to distinguish tuberculosis from lung plague in carcasses from which the pleuræ has been removed, *the chest skinned*, as the butchers say. (This gland has been called the motive (*motif*) gland of the inspector.)

Lydtin advocated his proposition as more practicable under present circumstances than Bouley's; while Bouley called for the *advocacy* of thorough measures, their *administration* was the concern of the authorities.

After further amendments of Rossignol and Van Hertsen, paragraph *f* of Lydtin was adopted, several members declining to vote.

Rossignol proposed to amend paragraph *g* by omitting the last sentence, permitting the use of the milk after boiling, and as thus amended it was adopted.

Bouley requested a vote on paragraph *h*, offering the following modification:

> It is proper to furnish an indemnity for cattle in good condition found to be tuberculous after slaughter.

Rossignol supported an indemnity for animals in good condition, but only when killed for human food.

Ærts, Wehenkel, and Lydtin opposed on the ground that this was only a question of goods of bad quality, the use of which for a purpose to which it is unfitted is forbidden. An indemnity should only be given when an owner must give up his chance of the recovery of a sick animal in the interest of the public good.

As a prolonged discussion was threatened and time pressed, Müller proposed the closure of the question which had already exceeded that proposed for the *order of the day*, namely, *the utilization of the flesh*. The discussion was accordingly closed.

A paper was handed in signed by eleven members explaining that they had declined to vote on tuberculosis on the ground of lack of time to sufficiently discuss such an important subject.

After providing for the next international veterinary congress to be held in Paris, the meeting adjourned.

REMARKS.

(The importance of the question of the sale of meat and milk from tuberculous animals cannot well be overrated. But to control the former we need a complete reformation of our system of slaughter in the large cities and villages. One municipal abattoir should be established in each great center of population where alone stock should be slaughtered for food and where the carcass and viscera of every animal slaughtered would be examined by a competent veterinary inspector. All establishments for the killing of meat to be shipped fresh to the cities, to be salted or canned, should be placed under similar supervision. Meat from uncontrolled slaughter-houses should be excluded. So far as we know no American city has adopted the system of municipal abattoir and complete veterinary inspection, and the great majority have neither. The expense would be considerable, though only a trifle as compared with that caused by the sicknesses, incapacity, and death now occurring from a disease which affects one fifth and upward of the population in the great cities. We say nothing of the other contagious diseases from which this measure would protect the people. With regard to the milk supply there should be frequent visitation of the dairies supplying the large cities, the maintenance of a census of the animals, and an inspection by a competent veterinarian of all cadavers of animals killed or dying by natural cause. Not only would this protect the human population against infection through the milk, but it would overcome the present great difficulty in dealing with the lung plague, which would thus be traced to every center of infection and could easily be stamped out. The question of the suppression of this disease over the entire national domain is by no means such an easy one; the task is so gigantic and the outlay would be so vast. In many herds in the Eastern States the proportion of tuberculous animals ranges from 10 to 30 per cent., and in the West with freer range it is doubtless far less frequent, yet if we were to estimate but one infected animal in thirty it would embrace over a million cattle and one and a half million hogs. Indemnities alone for this number would amount to from $30,000,000 to $40,000,000, to say nothing of all contingent expenses. That it would pay in the single item of the preservation of our live stock there can be no doubt, while its effect on the health of the population would be beyond all estimate. To the professional man, fully acquainted with the enormity of the evil, it is hard to wait for the slow growth of publico pinion while infancy and manhood are being cut down indiscriminately by a preventable contagion, and while even he himself must continually run the gauntlet of the deadly blow of the insatiate enemy. Every instinct of sanitation, of justice, of humanity, of self-preservation, and of morality rebels against the indifference of the people, but more particularly of the statesmen. But it is with tuberculosis for animals in general as with lung plague for cattle in particular, or with glanders for solipeds and man, the retribution for neglect is not executed with that promptitude which strikes terror to the mind; the laity fail to connect the final desolation with the distant cause, and the most deadly of all contagia is permitted to hold on its darksome way unchecked.

A single case of cholera, yellow fever, or even of small-pox in a large city is considered good cause for excited editorials in the newspapers and for extraordinary efforts on the part of boards of health, while every day from January to December scores are perishing unnecessarily from the more insidious and far more deadly tuberculosis.

The control of this affection cannot be advocated as either cheap or easy, or likely to be crowned by an early extinction of the disease as would be the case with lung plague. It will entail an immense organization, large expenditure, and persistent application, not only until all our flocks and herds are purified, but until the present tuberculous generation of men have given place to a healthier, and until by a slow improvement, generation by generation, the population shall have finally risen above this bane of our civilization. It is, however, a work that may be profitably undertaken by installments, first in the abattoirs and dairies of our large cities and suburbs, then in our markets for live stock, and our great emporia for butcher meat, then in all our large public institutions, and finally in our flocks and herds at large. It may safely be predicted for the city that will take the first effectual step in this direction that the showing made by her vital statistics will soon attest the wisdom of the course. The results will be no less beneficial to the stock owner, for apart from the protection of his own health and the assurance furnished him that he is providing a wholesome food for the people, his herds will be saved from a contagion which now causes incalculable losses over the entire country.

Upon our statesmen the burden of all this responsibility rests. They are intrusted with the expenditure of the nation's money, and with this trust they become severally responsible for every evil which it is in their power to ward off. To wait for the irresistible pressure of public opinion in a matter such as this, which appeals so strongly to their humanity, their religion, their patriotism, and self-preservation, is unworthy of the high place they hold and the charge which the people have confided to them. They have been chosen to do the work of legislation for which the people at large have no time; it is their duty to consider these matters when laid before them and to act on them, and on them must rest all the blame of the deaths and disasters that result from their apathy and neglect.)—J. L.

RESOLUTIONS ADOPTED.

First question.—Veterinary service.

1. To organize in each country a veterinary service, exclusively charged with all that pertains to this service, of which the members, all veterinarians, should be counselors of every department of the Government, but which should be more especially represented in close relation to the central power; that veterinary medicine should there have her chief of service.

2. The veterinary sanitary service should utilize the greatest possible number of veterinarians. It embraces the surveillance of fairs and markets of animals; the inspection of butcher meat and abattoirs; the control of rendering works; the inspection of breeding animals; the surveillance or direction of mutual assurance against the mortality of stock; the revision of the census lists of domestic animals, &c.; it comprehends the service of the state, and may be made international, embracing especially the repression and suppression of contagious and epizootic affections, also the control of the various other veterinary services.

3. Between the various states which, by a regular repressive and preventive service against epizootics, can furnish guarantees of a good veterinary sanitary police, there should be established a treaty having for its object—

(*a*) To apprise the other states as speedily as possible of any outbreak of rinderpest, pleuro-pneumonia, aphthous fever, sheep-pox, maladie du coit, glanders (or farcy), or of scab in sheep.

(*b*) To publish periodically a sanitary bulletin upon these maladies, their extent, progress, and extinction, which particulars should also be published in the international bulletin if judged necessary.

(*c*) To oppose these maladies by measures of sanitary police, which have first been discussed and adopted as the most desirable.

(*d*) To furnish with animals and herds, moved into or out of any territory, certificates of origin and health of a value guaranteed by the administration.

(*e*) To contribute to the publication of an international veterinary sanitary bulletin.

Second question.—Pleuro-pneumonia.—Lung plague.

A.—DIFFERENTIAL DIAGNOSIS.

1. From an anatomical point of view, at least in its relation to veterinary police, one may consider as epizootic contagiou[illegible]leuro-pneu-

monia; every pneumonia which is lobular and at the same time interlobular, and the development of which does not depend on traumatic causes.

2. From a physiological standpoint in the living animal, epizootic contagious pleuro-pneumonia is specially characterized by its contagious character and by the symptoms of lobular pneumonia.

3. In an infected place every animal which shows febrile reaction or symptoms of disease of the chest should be considered as suspected of pleuro-pneumonia. Every animal found in an infected stable or which has been in one within three months antecedent, or which has been able to be contaminated in any other way, should be considered as suspected of contamination.

B.—PROPHYLAXIS.

1. Recognizing that from the point of view of sanitary police epizootic pleuro-pneumonia is a disease which propagates itself only by contagion, and is usually incurable and fatal, the congress declared that to prevent the development and propagation of this malady there should be applied the measures against contagious diseases that are at the same time fatal and incurable.

2. Animals diseased or suspected of the disease should be sacrificed as quickly as possible.

3. Contaminated animals or those very much exposed to the contagion should be isolated or sacrificed. The slaughter of contaminated animals is especially indicated when the disease manifests itself very exceptionally, or for the first time in a stable belonging to a commune or country rich in cattle.

4. We have to-day experimental proof that it is possible to invest the organism of animals of the horned species with an immunity from contagious pleuro-pneumonia by inoculation with the virus of this malady.

5. Preventive inoculation, that is to say, that which is practiced when the malady does not prevail in a country, ought to be absolutely rejected. Inoculation, so called, of necessity, that is to say, that which is practiced when the disease exists in a herd, may be permitted but not made obligatory.

6. The inoculation should always be done by a veterinarian.

7. It is not proved that an inoculated animal cannot transmit the disease to a sound animal.

8. Inoculated animals should be reported to the authorities.

9. Quarantined animals should be made the object of a special census, and should receive a distinctive brand with a hot iron.

10. No animal suspected of infection should be moved without previous authorization of the communal administration. The permit to move should only be granted for animals destined to the butchery; it should only take place in special conditions under the supervision of the police, and in such a manner as to prevent all propagation of the malady.

11. Every bovine animal offered for sale ought to be accompanied by a certificate of health testifying that no epizootic has existed for at least six weeks in the commune from which it came.

12. In certain special cases, to be determined by the official veterinarian, there may be prescribed the suspension of fairs and markets, the prohibition of the importation of animals from a suspected country, quarantine, the posting of notices at the entrance of infected circles or farms, and the issue of handbills and instructions warning the people of their obligations, and of the precautions to be taken to prevent the appearance or extension of the malady.

13. An active supervision should be exercised, not only over the quarantined animals, but also, *a*, over animals exposed for sale in markets, fields, and fairs; *b*, over animals lodged temporarily in the stables of inns adjoining the markets; *c*, in stables containing many animals, subject to frequent changes, and when the malady has already made one or more outbreaks.

14. The duration of quarantine should be six months at least, apart from the last case of the malady.

15. At the release from quarantine the animals should receive a second mark to annul the effect of the first.

16. The flesh of an animal slaughtered should not be used for food, unless authorized by the veterinarian making the autopsy.

17. The skin should not be utilized until it has been steeped for twenty-four hours in an approved disinfectant solution.

18. Carcasses and cadaveric *débris*, unfit for food, should be buried, or so treated as to be absolutely inoffensive.

19. Stables, fair grounds, markets, and vehicles which have been occupied by diseased or suspected animals, should be carefully cleansed, disinfected, and purified. These different operations should be under the direction of a veterinarian.

20. A stable should not be refilled with animals until it has been entirely evacuated and thoroughly disinfected, and then purified by eight days of free ventilation.

21. Pastures that have been occupied by diseased animals ought to be quarantined for at least fifteen days.

22. The different materials, objects, and instruments that have been employed in the slaughter, transportation, or burial of diseased or suspected animals, should be destroyed, or thoroughly disinfected. Forage and litter should be utilized for horses or other solipeds.

23. Persons who have become soiled by infecting materials, should wash their hands, wash or brush their clothes, and wash their boots with a disinfectant solution.

24. All persons and animals capable of carrying the virus should as far as possible be kept from diseased animals, their carcasses and cadaveric débris.

25. It is proper to grant an indemnity to owners for animals slaughtered by official order and for the cost of disinfection. The indemnity should amount to four-fifths of the value of the animal, and to the full value, deduction being made of the value of portions of the carcass that can be utilized if the animal should prove healthy.

26. Very heavy penalties should be imposed on persons who violate the different sanitary regulations decreed by the authorities.

27. A good organization of the veterinary service is the best guarantee of the application of the different measures prescribed.

28. A last and potent measure for securing the extinction of contagious pleuro-pneumonia would be the adoption of means for the contagious diseases of animals similar to that in use for the phyloxera of the vine; to formulate an international agreement in which shall be indicated the essential elements that ought to form the basis of legislation to be adopted by each country which shall join to carry it out.

Third question—Veterinary education.

1. To be admitted to veterinary studies one must be batchelor es-lettres or es-sciences; that is to say, he must have finished the studies of the secondary education.

2. There is no call to create veterinarians of different classes having a different amount of preparatory or veterinary education.

3. Four years of study at least are requisite to make a full study of veterinary medicine, if that is made to include physics and natural sciences.

(*a*) The instruction of the two first years (four first semesters) should embrace the following branches: physics, chemistry, natural history (zoology, mineralogy, botany, and geology), anatomy, histology, physiology, and shoeing. A course of practice and demonstrations in micrography should always be included.

(*b*) Clinical teaching should continue through the whole of the last two years of study. That the practical instruction of the student may be complete it is absolutely necessary to have, beside a stationary and consulting clinic (hospital clinic and polyclinic), an ambulatory clinic (outside clinic); there ought to be at least two professors of clinics.

(*c*) The inspection of butcher-meat is an absolutely essential branch of veterinary education.

4. At the end of each year veterinary students should be examined on the studies which they have been taught that year. No one should be allowed to follow the course of the advanced year unless he has passed this examination.

No one should be admitted to examination for the degree of veterinarian who has not followed a course of clinical instruction for two years after having passed the examination of the second year of study.

The board of examiners for granting degrees should always be formed partly of professors and partly of practitioners.

5. Internal (board under military rule) and external (residence outside) are optional in veterinary schools.

6. Veterinary schools may be independent establishments, or they may be connected with universities or institutions for the higher education, but veterinary medicine should have its special chairs. One cannot but disapprove of institutions in which all branches of veterinary education are divided in a very limited number of veterinary chairs; such a system is absolutely insufficient.

It is very desirable that in every country veterinary schools should be state institutions.

7. The professors of veterinary schools ought to possess diplomas of veterinary medicine. It is very desirable that veterinarians before being called to the professorship should have practiced veterinary medicine for some years.

Fourth question.

(See body of report.)

Fifth question—Tuberculosis.

The flesh and viscera of a tuberculous animal can only be utilized for human food when the disease is found in the cadaver in the incipient stage, when the lesions are confined to a very small portion of the body, when the lymphatic glands are still free from all morbid tubercular lesions, when the tuberculous formations have not yet undergone softening, when the flesh presents the characters of meat of the first quality, and when the animal is in good state of nutrition at the time of slaughter.

It should not be permitted to remove the flesh of tuberculous animals admitted to consumption out of the locality where they have been slaughtered, and it should not be offered for sale in the butchers' stall. Every quarter of meat and all viscera showing lesions of tuberculosis, as well as the flesh of any other animal in which there is found at the necropsy a tuberculous infection more pronounced than that referred to above, should be watered with petroleum oil, and afterward buried under police supervision. The extraction of fat by cooking and the utilization of the skin may be permitted.

The inspection of every animal attacked by tuberculosis should be made by a veterinarian, who alone should decide if the flesh is fit for human consumption.

The milk of animals affected with tuberculosis or suspected of it should not be taken by man nor by certain animals. The sale of this milk should be severely interdicted.

Respectfully submitted.

JAMES LAW.

Hon. GEORGE B. LORING,
Commissioner of Agriculture.

HAMBURG INTERNATIONAL EXHIBITION.

REPORT OF J. H. SANDERS.

Hon. GEO. B. LORING,
Commissioner of Agriculture.

SIR: On the 19th day of May, 1883, I had the honor to receive an appointment from you with the following instructions:

> You are hereby appointed and commissioned an agent of the United States Department of Agriculture to attend the International Exhibition at Hamburg in 1883, and you are instructed to examine and report upon the following special points:
>
> The character of the Exhibition in all its departments of live stock as compared with our own.
>
> The general condition and supply of live stock in England and on the Continent.
>
> The breeds most met with and that seem to be most desirable and popular; also, their relative merits as compared with our own.
>
> The probability of improvement to our own stock by further importations, and the breeds most desirable for this purpose, if any.
>
> The dangers from contagious diseases that may be apprehended from continued importations, if any.
>
> The methods of live stock husbandry practised compared with our own.
>
> The probable future demand from European countries for American live stock and meats.
>
> The present hindrances and restrictions that affect the exportation of live stock from the United States, and how they may be modified or removed, especially with reference to the exportation of our live cattle to Great Britain, and our hog products to Germany.

In pursuance of the foregoing instructions, I started upon my mission May 26, landing at Liverpool. After a brief sojourn in that city, devoted mainly to an examination of its cart-horses, I proceeded to London, where I spent several days familiarizing myself with the markets, the modes of travel, and other matters of importance connected with the live-stock industries of that city. As one of the principal objective points of my visit was the International Live Stock Show at Hamburg, I determined to go at once to the continent, leaving England and Scotland for my return trip. Accordingly, June 19, I left London purposing to make my journey to Hamburg via Paris, stopping *en route* to make a study of

THE DRAFT HORSES OF FRANCE.

The constantly increasing popularity of the French draft horses in this country, as evinced by the large numbers annually imported, and the interest I myself had taken in some of the discussions growing out of the business, led me to attach a good deal of importance to what I might learn concerning them by a careful personal investigation of some of the controverted points. Among the most important of these were the following:

1st. Are there any well-established distinct breeds of draft-horses in France? 2d. What guarantees, if any, can American importers obtain in France that the horses purchased by them belong to any particular race or breed, and that they are purely bred? And 3d. To what breed do the horses usually brought to America from France belong, and in what part of France are they bred?

It may not be out of place for me to remark here that during the past six years I have, myself, been compelled to participate in several controversies, through the agricultural and live-stock papers of the country, touching the draft horses of France and the name by which they should be known in the United States. Throughout these controversies I relied mainly upon such of the printed history of these horses as had been translated into English, and upon what I could gather from intelligent gentlemen who had personally given more or less attention to the subject in France. It was therefore with a good deal of pleasure and interest that I embraced the opportunity afforded me, by my present visit, to go into this question thoroughly for myself. And first let me say that many American importers, especially the earlier ones, and some even at the present day, go to Paris, buy their horses of a dealer who has gathered them up from various quarters, and then return without the slightest knowledge of the breeding of their horses, or of the region where they are bred. It is not to be wondered at, therefore, that such men can tell nothing of the breeding of these horses when they get home, because they know nothing, beyond the fact that they had bought them of a French dealer at Paris, Dieppe, or Havre. If they attempted to say anything beyond this, they simply drew on their imagination for their so-called facts. After awhile, however, those with some experience in the business, and with some knowledge of good horses, began to inquire into the subject, to try to find out where the *good* horses came from, so that they might get them, if possible, from first hands. I began my investigations in the same way. I went first to the sales stables of Paris, fortified with all the knowledge I had been able previously to obtain upon the subject. I went first to M. Vidal, who is a noted horse dealer of Paris, and who has undoubtedly sold more stallions to American importers than all the other horse dealers of Paris combined. In reply to my question, he said: "Fully 90 per cent. of the horses that I buy to sell to Americans, for stallions, come from beyond Chartres, in the Perche; the others are picked up here and there, wherever we can find one good enough for the market; but we sell them all as Percherons." The other dealers all told substantially the same story.

After spending a few days in Paris, talking with horsemen and gathering what information I could, I determined to see the Percheron breeding district for myself. All authorities agreed in pointing out Nogent le Rotrou, situated about 100 miles southwest from Paris, in the ancient province of La Perche, as the heart of the Percheron breeding country. It is at and near this place that Mr. Dunham has bought hundreds of horses within the past three years for the American trade, and where the Messrs. Dillon and several of the other importers have made most of their purchases for some time past; and in this region is the only place where any attempt is made to maintain the Percheron race in its purity.

On the day of my arrival at Nogent le Rotrou, a large number of Percheron breeders had met to consult upon the propriety of establishing a Percheron stud-book, in order to preserve the purity of the race and to protect themselves from unscrupulous dealers in Paris and elsewhere. I was much interested in the discussion which took place. The gentlemen present represented the principal breeders for some 20 or 30 miles around, and I was told that they owned at least 100 stallions that had been kept for service this season. I questioned many of them. Among others, the statement of Mr. Ernst Perriot, one of the most noted of the breeders present, is a fair sample of what all had to say. He is a very intelligent gentleman, and has sold many horses to American buyers. I should judge him to be about fifty years of age. His statement was in substance as follows:

> I have been breeding horses right here all my life, and my father and grandfather were in the same business before me. We never breed or sell any other than pure Percherons. We have usually kept 6 or 7 stallions each year for service. They travel around the country, serving mares owned by the farmers at about 25 francs each. We keep an eye on these mares, know where the best ones are, and when the foals are weaned we buy many of the best ones each year and keep them until we can sell them at a fair profit. I am sure there has been nothing but recognized pure Percheron stallions used in our stud since the time of my grandfather, and nothing else has been used in this whole Percheron region within my knowledge. There is a tradition that about the time my grandfather engaged in the business some Boullonais blood was introduced into this country for the purpose of increasing the size of the Percherons, but certainly there has been none since about fifty years ago. The true Percherons will now average as large or larger than the Boullonais. Neither Mr. Dunham, Mr. Dillon, nor any other American importer has ever bought any Norman horses here. We don't have any such horses. You can see plenty of Normans in Paris; they are all small horses, mostly bays, and are used in the cabs and carriages. They are generally half-bloods got by English thoroughbred sires, and some of them are out of Percheron dams, and are usually called Anglo-Normans.

The same statements, substantially, in regard to purity of race and the name, were obtained from all the breeders interviewed, notably Auguste Tacheau (province of Sarthe), Pierre Sagot (province of Eure-et-Loir), and Celestin Caget (province of Orne). In fact, so far as I could learn, it was almost an insult to ask one of the breeders present if they bred or sold any other than Percheron horses, and they spurned the term "Norman" with contempt.

These are the men of whom all the leading American importers have been buying for several years past. They buy these horses as Percherons, from men who will call them nothing else, and yet a few of them come home and abuse everybody who refuses to call them Normans. There is not much in a name, but sometimes a name contains a deal of meaning within itself.

It was an agreeable surprise to me to learn that so much pains had been taken by these Percherons breeders to preserve the purity of the race. It had been asserted that nobody knew anything about it; that the Percherons were mongrels, and that no man could give the pedigree of his horse. I found, on the contrary, that, while they have not paid much attention to preserving the maternal genealogy, many of the sires can easily be traced six or eight generations. One young horse, now owned by Mr. Ernst Perriot, is a noteworthy example. This horse is a three-year old, and is so highly valued by his owner that he has absolutely refused to name a price on him. Fifteen thousand francs were offered for him by an American buyer in my hearing; but the owner shook his head and said, "You might safely double it." This young horse was got by the black horse Brilliant (No. 1271 of the Percheron-Norman Stud Book), that now stands at the head of Mr. Dunham's stud, at Wayne, Ill., and the line of sires back of him is known for seven generations. Mr. Perriot proposes to raise the service fee of this horse to 100 francs next season, a price that has hitherto been unheard of in the Percheron breeding district. As another instance of the care which French breeders are taking to secure good breeding stock for themselves I may mention the fact that Mr. Auguste Tacheau recently paid 3,000 francs for a suckling colt, which he designs to use in the stud.

You will doubtless remember that one of the things discussed between us at our interview a few days prior to my departure upon this mission was the desirability of a Percheron stud-book in France in order to protect American breeders from imposition, and to afford some guarantee of the purity of the blood of the horses brought to this country from France. And you will also no doubt remember that the desirability of such a publication was made the subject of a special communication from you to the French minister of agriculture, at that time. It is therefore with no small degree of satisfaction that I am able to report that the Percheron breeders of France have fully resolved on a stud-book. The meeting to which I have heretofore alluded, as having occurred on the day of my arrival at Nogent le Rotrou, was simply informal and preliminary, but on Saturday, June 23, a largely attended meeting was held at the same place, about one hundred breeders, representing the provinces of Eure-et-Loir, Orne, Sarthe, and the whole of the ancient province of La Perche, being present. At this meeting a permanent organization was effected under the sanction of the Government, with M. Fardouet as president, and Messrs. Vinault, Perriot de Champeau, Sagot Anatote Miard, Poullain, Lucas, Launay, Aveline, Tacheau, and Caget as vice-presidents; M. Boullay-Chaumard, secretary-treasurer. These, with the mayor of the city of Nogent le Rotrou, the prefect of the department of Eure-et-Loir, and the prefect of Nogent le Rotrou, constitute the "board of direction" of the Société Hippique Percheronne. The rules for registration are very strict, and every application must be sworn to, giving the genealogy as far as it can be traced, and establishing beyond controversy that the animal is "born Percheronne." Penalties are provided to prevent fraudulent entries or disreputable transactions generally. In short, it looks like a strong, resolute, aggressive organization from the start, and will mark an era in the Percheron breeding business.

I conclude, from a considerable sojourn in France, that Americans who buy directly from the breeders and "stallioners" (stallion keepers) of the Percheron district have heretofore had a reasonably certain guarantee of purity of blood, and henceforth the assurance will be as complete as can be obtained with any breed of draft horses in the world. Those who buy of the dealers in Paris and in the cities on the coast can have no such assurance, and their own eyes must judge of the breeding It is to be hoped that hereafter no imported horses will be admitted to registry in the Percheron-Norman stud-book of America that are not previously registered in the Percheron Stud-Book of France. It would have been infinitely better for the reputation of the breed, and for our own horse stock, if such a regulation had been practicable and enforced years ago.

There are many most excellent horses working in the drays, trucks, and omnibuses of Paris. The omnibus horses will average much larger than has been represented by most writers. They are usually thin in flesh, and the weights, when given at all, have been with reference to that condition. I stood on the street one day for an hour in company with two experienced importers, watching these horses, especially with a view to estimating the average size. The verdict was: average weight in good flesh—fat as stallions are usually kept in America—between 1,400 and 1,500 pounds. These omnibuses are enormous, great double-deckers, holding about sixty-five persons, and are drawn by three horses harnessed abreast. Fully seventy-five per cent. of these horses are white, and they nearly all have the usual Percheron characteristics. M. Vidal, the Parisian horse dealer previously alluded to, told me that he sold about 1,100 horses a year for use in the omnibuses; that nearly all of them came from beyond Chartres in the Perche, and that the average weight was about 1,400 pounds. The horses one sees in the drays are larger; an average lot of imported Percheron horses is but little if any better or larger than the average cart-horse as seen in the streets of Paris. They are massive, strong, patient fellows, and are nearly all Percherons.

In no city that I have visited are the horses driven so hard and treated so unmercifully as in Paris. The drivers whip their horses and rush about like madmen, and although the streets are very wide and rarely crowded, yet the pedestrian who attempts to cross must keep a sharp lookout or he will be run over. The cab-horses of this city are a sorry, rough looking lot, which I attribute mainly to their bad usage. The drivers of the cart-horses do not seem to be quite so unmerciful, but throughout all Paris the prevalent idea with a driver seems to be, that a horse was made to *use* and to *abuse*. Even in the Percheron breeding districts I noticed traces of this same feeling, and here also I noticed the most striking lack of a knowledge of the value of cleanliness in the management of a horse. The French are miserable groomers; their horses are filthy and dirty, and are left mainly to take care of themselves, so far as cleanliness is concerned.

THE HAMBURG INTERNATIONAL EXPOSITION.

From Paris I went direct to Hamburg to visit the exposition, or "International Show," as it was called, which opened July 2. It was in many respects a disappointment. In some features it was equal to an ordinary Ohio, Illinois, or Iowa State Fair, but in many others it was far behind the usual annual exhibit in those states. In the single feature of dairy stock it was great; and I never had so high an appreciation of the quality of the Dutch-Friesian or Holstein cattle as since seeing them at this show. It is, however, so far as I can learn, the best exhibition of the kind ever held on the continent, the number of entries largely exceeding those of the Paris Exposition of 1878.

The "classes" were legion. There were 56 classes for horses and mules, with 424 entries; 141 classes for cattle, with 902 entries; 88 classes for sheep, with 728 entries; and 50 classes for pigs, with 277 entries—certainly classes enough and entries enough to make a grand show. But, unfortunately, many of the entry numbers were blank, a condition of things which seems inseparable from the system of numbering and cataloguing an exhibition of this nature in advance of the opening.

The various breeds of live stock of Germany and of Northern Europe generally were well represented, with the exception of those of Russia, from which country I was not able to find anything. In this I was disappointed, as I had hoped to see a good representation of the famous Orloff trotting horses. The show of draft-horses was scarcely worth mentioning. There were a few creditable specimens of the English cart-horse, and some draft-horses from Belgium, but none worthy of especial notice. The government breeding studs, of Germany, were pretty well represented with horses of the thoroughbred type; very fair specimens, but in no wise remarkable. In short, the horse-exhibit generally impressed me as a heterogeneous mass of no especial excellence in any particular. There were some dun ponies, from Norway, about 14 1-2 hands high, very heavily built, and blocky—enormous draft horses in miniature, that were much admired. There were no French horses on exhibition. In fact no stock from France, so far as I could learn, except French merino sheep.

The feature of the cattle show was the dairy stock, and in this I never saw anything that could approach it. Hundreds of the Friesian or Holstein cows were there, with their grand udders, and every indication of milk-productng capacity of the highest order. I could not help remarking to some American friends who were with me that I feared our importers had not yet taken across the waters any of the *best* of the race, for I certainly never saw such dairy cows in any show-yard in America as I saw at Hamburg. I am now more than ever convinced that, all things considered, the Dutch cow is the general dairy cow, *par excellence*, of the world.

I was also very much interested in the Oldenburg cattle, a race that in color closely resembles the Holstein or Friesian breed; rather darker in color, not quite so good, I should say, for the dairy, but very much better for beef. In fact, the very best beef cattle on exhibition, in my opinion, were these same Oldenburgers. They are smooth, rather fine in bone, handle fairly well, and bear every indication of good feeding quality. If it were possible to combine the beef-making quality of the Oldenburg cattle with the great milking capacity of the Dutch cows it would come very near making the model cow for the average farmer.

Another class of cattle that interested me much was the Angeler, a race that seems to be peculiar to Schleswig-Holstein; little fellows, rather smaller than the average Jersey; very dark red, bordering on brown in color, very fine in the bone, and evidently deep and very rich milkers. Mr. H. Chandos-Pole-Gell, one of the delegates from the Royal Agricultural Society of England, remarked to me that to him this class was the most interesting one in the show; but while I freely concede to them great milking and butter capacity in proportion to size, I could not help thinking of "Uncle Billy Smith's" *bon mot*, anent the Jerseys, at the Illinois State Fair two years ago: "They will do for a man who is too poor to keep a cow and who is ashamed to keep a goat." They are *too little* to be of much practical use, but they are evidently a very pure race, and good machines for making milk and cream, in proportion to their size.

There were some short-horns on exhibition, but they were, with the possible exception of one or two cows, miserable specimens of the breed. There were also a few Ayrshires, and one or two Polled-Angus, of very ordinary merit. The Swiss cattle were out in considerable force, and, from their uniform gray color and the immense bells that accompanied them, attracted a good deal of attention. They were of fair size, pretty good on the back, many of them, but coarse and bad handlers, evidently lacking in what cattle breeders usually denominate "quality."

As a matter of curiosity I was interested in the little cows from Norway. They are little things, much smaller than the Angeler cows above alluded to, with remarkably thin, flat bodies, on very short legs, white on back and belly, the sides a light yellow, streaked with black, marking them much like a tiger. About a dozen of them were in the show.

The show of sheep was not remarkable in any feature. The French merinos rather took the lead in numbers, closely followed by the English mutton breeds, the various classes of Downs, with a few Cotswolds and Lincolns. Of the French merinos, there were many most excellent specimens, better and larger than any I had heretofore seen. The people of Germany appear to be "taking to" the Southdowns, Shropshires, Hampshires, and Oxfords largely.

In the swine department of the show I was pleased to see our American-Poland Chinas holding a prominent place, and, in my judgment, the very best specimens of swine on exhibition were of this breed and imported from America. Mr. John C. Funch, of Oldenburg, was the enterprising gentleman who made this exhibit, and his swine are certainly most creditable representatives of the breed. In point of numbers, the white breeds, mostly of the various Yorkshire families, made up about 60 per cent. of the show, while the remainder were about equally divided between the Berkshires and Poland-Chinas, with a few Essex.

I may sum up my impressions of this show by saying that, while it was very creditable in most departments and great in display of dairy stock, I saw nothing worthy of especial remark in the management. The system of classification was very complete, and the several breeds were usually grouped together in their stalls or pens so that one desiring to examine any class of stock found but little difficulty in doing so. The published catalogue of the exhibits was also a great convenience to visitors, as in it full particulars as to name, age, pedigree, ownership, etc., were given. The custom of issuing a catalogue of this sort is general with European agricultural shows, and might be followed in our own country with profit.

I saw no breeds of live stock at this show the importation of which to the United States, in my judgment, should be encouraged or recommended other than the Holstein or Friesian-Dutch dairy cattle and the Oldenburgs, to which I have alluded in the foregoing. They are certainly most excellent breeds of cattle for the purposes indicated. The former have already been extensively introduced in our country, and are certainly rapidly growing in public favor. To what extent the Oldenburgs might be profitably imported, if at all, I am not prepared to say. I can only reiterate my former statement, that they were certainly the best beef animals on exhibition, and that they also seem to possess an adaptation to the uses of the general dairy farmer, but little inferior to the Holsteins or Friesians, which they closely resemble.

OUR PORK IN GERMANY.

While in Germany I made diligent inquiry in order to ascertain so far as possible the feeling of the Government and people concerning the admission of American pork to that country. Among the trades people in the cities, so far as I could learn, the feeling is very strong against the prohibitory policy of the Government, but among the best informed people, and those connected with the Government, the case is just about as follows:

The swine of both Germany and the United States are confessedly, to some extent, infected with trichina. The German authorities require that the flesh of all swine slaughtered in that country shall be subjected to a microscopic examination by a Government inspector, and pronounced free from trichina, before it can be offered for sale. No such examination is practicable with salted and packed meats from America, and as no pretense of an inspection of our meats is made by us under Government authority, or even under the supervision of the packing firms, the German Government says, "We cannot accord to American packers and butchers a privilege which we refuse to accord to our own." So far as I could gather, it is generally conceded that whenever an inspection of our pork is made, under careful governmental supervision, similar to that now made by the German authorities, there will be no objection to its sale in that country. Indeed it strikes me that were such an inspection made there would be such a strong pressure brought to bear upon the German Government by their own people that the embargo would be speedily removed. The presence or absence of this microscopic parasite in the pork of this country is a matter of but little interest to us, as Americans *cook* their pork before eating it, and thorough cooking obviates all danger, but in Germany, where much of it is eaten raw, it becomes a serious matter. Should further experiments confirm what has been already partially shown, that thorough curing in salt destroys the vitality of the trichina, if not entirely, at least, to so great a degree as to reduce the danger from meats that have been thoroughly cured by this process to almost nothing, it will doubtless be a strong point in our favor, and a proper presentation of the facts to the German Government, should this be clearly proven, ought at once to be made. Until this fact is clearly established, however, and in the absence of any pretence of inspection of American meats, either under governmental or individual supervision, the position of the German Government is obviously sound upon this question, and cannot be assailed without first demonstrating that their own inspection is useless.

OUR EXPORT CATTLE TRADE WITH GREAT BRITAIN.

From all that I could learn while in England, the trade in dead meat between the United States and Great Britain is not at all satisfactory either to those engaged in the trade or to the consumer. The facilities for securing the exact temperature necessary in order to maintain the proper condition of the meat during the ocean voyage are so imperfect that the meat not unfrequently reaches the consumer in a more or less damaged condition, and large losses have resulted. All well-informed persons agree that the most satisfactory results will be attained when we shall once more be able to show a clean bill of health, and send our live cattle, under such regulations as will insure their health and comfort, to the farms and pastures of England and Scotland, there to be slaughtered when their condition and the state of the market requires it.

It ought not to be considered strange that British farmers should make strenuous efforts to still further restrict or totally suppress the importation of live stock for slaughter in their country. They have suffered enormous losses in times past from imported disease, and during the last few years they have been contending with great obstacles, not the least of which has been the great supply of agricultural products that America has sent to that country. Under these circumstances it is not to be expected that British farmers will be inclined to take a strictly judicial view of the case. Naturally they would like to secure a better market for their own stock, and equally naturally they magnify possible danger of importing disease from this country. On the other hand, it should be borne in mind that the English Government has steadily refused to yield to the demands for further restrictions, and has in the main done full justice to American interests, especially in the recent alarm over the reported existence of foot-and-mouth disease in this country.

It is doubtless true that British farmers do not regard the existing condition of the meat trade with this country favorably, and that whenever they are satisfied that it may be done with safety they would much prefer that the restriction should be removed altogether, rather than that the present arrangement of compulsory slaughter be continued. As the trade is now conducted the supply received from the

United States detracts just so much from the demand for home products, and the British farmer has no opportunity for even an incidental share in the profits of the business. On the other hand, could our cattle be safely admitted without restrictions, immense numbers of store or partially fatted cattle would be shipped to that country, and would be taken inland to British pastures and finished up on British food, thus enabling British farmers to reap profits out of the better fitting of these cattle for the market, and also to utilize them in the converting of the provender of the farm into manure, a very important consideration with the farmers of that country. During my visit to that country I conversed with many farmers upon this subject, and I never met with a single man who expressed himself as otherwise than favorably disposed towards the free admission of American cattle whenever it could be done with a reasonable guarantee of safety from contagious diseases; and in nearly every case the hope was expressed that the time would soon come when the present restrictions might safely be removed. Instead of being opposed from motives of self-interest to the free introduction of our cattle to that country, the feeling seems to be general among English farmers, so far as I heard any expression of opinion, that the profit derived from the "finishing up" process would more than compensate for the resulting competition.

Feeling certain that this is the view generally held by intelligent and progressive British farmers, and also strong in the belief that we shall at no very distant day be in such a condition as to enable us to give our British cousins assurances concerning the sanitary condition of our cattle that will be satisfactory, I confidently look to the entire removal of the present restrictions as a thing reasonably certain to occur in the near future. Of course this belief and hope is based upon the confidence I feel in the course which our General Government, aided by that of the infected States, will take in respect to the stamping out of contagious diseases and in otherwise improving the sanitary condition of our cattle, and also in the surveillance which, in my opinion, will at no distant day be exercised over our exports.

The importation of cattle from foreign countries was a fruitful theme for discussion in Great Britain during the time I was in that country. The unusual extent to which foot-and-mouth disease has prevailed there during the past year and the losses resulting therefrom to British farmers have made them extremely restive, and while the Government has been active in its efforts to stamp out the disease, it has been constantly importuned to adopt still more stringent regulations to prevent the introduction of contagious diseases from abroad. All Americans interested in the subject are aware that for several years past a regulation has been enforced which requires that all cattle brought to Great Britain from the United States must be slaughtered at the docks where landed within ten days after their arrival, no matter what may be their condition or the state of the market. This edict of compulsory slaughter is based upon the assumption that to admit the free transit of cattle from the United States to the farms and pastures of Great Britain would expose the cattle of British farmers to increased dangers of infection from pleuro-pneumonia or lung plague, the presence of which disease along a portion of our Atlantic seaboard is admitted.

In the mean time Canadian cattle have been freely admitted without compulsory slaughter or detention, no contagious diseases having been found among the cattle of that country, and the Canadian Government maintaining a rigid quarantine against the cattle of all foreign countries, the United States included, and also making a careful inspection of all exported cattle and exercising a rigid supervision over the ships in which they are carried. In consequence of these regulations a Canadian bullock will bring $15 to $25 more at the Liverpool or London landings than could be obtained for the same bullock if exported from the United States. To some European countries where the sanitary condition of the cattle is not so satisfactory as in the United States even the grace of compulsory slaughter is not accorded, but the landing of live cattle therefrom is absolutely prohibited.

In January last a shipment of cattle was received at Liverpool from Boston, which, upon inspection, was found to be suffering from foot-and-mouth disease. In March a shipment was received from Baltimore in the same condition. The publication of these facts led to a strong popular demand upon the Government that absolute prohibition should be applied to all cattle from the United States; and in July, while I was in London, Mr. Chaplin introduced a motion in the House of Commons which was, to all intents and purposes, a demand upon the Government that such action should be immediately taken. In spite of the opposition of the Government party, Mr. Chaplin's motion prevailed by a majority of eight.

I listened with a good deal of interest to the discussion which this motion elicited, and, in common with all other Americans who took any interest whatever in the subject, I regarded the vote by which it was carried as a very threatening omen. Indeed, the agent of the Dominion Government of Canada said to me the day after this vote was taken that he was certain that within a week an order would be issued absolutely prohibiting the landing of American cattle at British ports. I set at once actively to do what I could to counteract this. The pretext set up for this action was the presence of foot-and-mouth disease in the United States. My position as an accredited representative of our Department of Agriculture, and my opportunities for ascertaining the facts as a member of the Treasury Cattle Commission, enabled me to make representations touching the actual condition of our cattle that attracted attention, and which I believe were quite generally accepted as true. Through the London Times and by personal interviews with members of Parliament and with the chief veterinary adviser of the privy council, aided, I may say, by the efforts of others, I was able not only to counteract the effect of the vote on Mr. Chaplin's motion, but to bring about a much more satisfactory state of feeling, so far as the security of the present condition of the trade is concerned, than had previously existed.

I was able to explicitly deny the existence of foot-and-mouth disease in the United States, except in cases of direct importations from Great Britain, which cases had been promptly detected and closely quarantined, and to state positively that the cases of foot-and-mouth disease alleged to have been brought from the United States were due solely to the infected ships which had brought diseased cattle to our own ports, and without proper disinfection had been loaded with fat cattle for Liverpool.

The effect produced by this plain statement of facts was so considerable that Mr. Arnold called attention in the House of Commons to the assurances contained in my letter in the Times of July 18th, and also to one of a subsequent date from General Carman, of the Department of Agriculture at Washington, and asked the Government: "Whether, in view of these facts, the present restrictions might not safely be removed so far as cattle from the Western States were concerned."

The answer from the Government was, as I supposed it would be, to the effect that, under the existing act of Parliament, no concession could be made in favor of a portion of any country that could not be accorded to it as a whole. I succeeded in obtaining from Professor Brown, the chief veterinary adviser of the privy council, an admission that in his opinion a plan might be devised by which cattle from our Western States might now be admitted with reasonable safety. This I regarded as a very important admission; but the nearness of the end of the session of Parliament made it useless then to ask for such additional legislation as would be necessary in order to enable the privy council to carry out this idea. I may add, in this connection, that Professor Brown, speaking for the privy council, expressed himself as greatly pleased with the steps which the Agricultural and the Treasury Departments of the United States had taken within the past year or two to improve the sanitary condition of our live stock and to prevent the importation and spread of contagious diseases; and that the impression is rapidly gaining ground that we shall soon be in such a condition that cattle from all parts of the United States may be admitted and taken inland without any danger from disease. This feeling is based mainly upon the steps which our Government has already taken, and a faith that we shall continue to go forward in the same direction.

I cannot but regard it as extremely fortunate that some one who could speak with some degree of assurance, and whose opportunities, from knowing the facts, were acknowledged, should have been on the spot n London in July last to correct the misapprehensions that existed touching the prevalence of foot-and-mouth disease in this country; and I think it reasonably safe to predict that, with the enforcement of such regulations as are now contemplated by our Treasury Department, and with such additional legislation as may be reasonably expected from our Congress at its next session, the time is not far distant when American cattle will be placed upon an equal footing with those from Canada so far as admission to British ports is concerned.

IMPRESSIONS OF BRITISH BREEDS OF CATTLE.

However much the substantial merits of short-horns in Great Britain, as well as in America, may have been damaged within the past twenty years by blind allegiance to what has been called "fashionable breeding," it is evident that the "color craze," which prevails to so great an extent in the United States, has not yet had any serious effect on the other side of the ocean. In the stables, the pastures, and the show-yards of England and Scotland I saw comparatively few red short-horns. Roan appears to be the favorite color there; the all-white being quite as frequently met with as the all-red. In the show-yards the rich red-roan—a color that is peculiarly characteristic of the short-horn breed—appears to be in a decided majority over all others.

In point of real merit, however, I think no candid man who has seen the cattle of both countries will dispute my assertion when I say that, judged by the cattle shown at the Royal this year, English short-horns are not equal to those shown at our leading fairs in America. There were no representatives of the breed at the show of the Royal Agricultural Society, at York, this year, worthy of being compared with the show herds that Potts, Pickrell, Sodowsky, Croft, and Palmer exhibited throughout the Western States at our principal fairs last year. In this opinion I am sustained by every American "cattle-man" that I met at York during the Royal show, and there were several of them—Hereford, Angus, and Devon, as well as short-horn breeders. With the exception of the yearling and the two-year-old heifer classes, I thought the show weak throughout, so far as quality is concerned, although it was quite strong in numbers. At this writing I have not read any of the comments upon this show by the English press, so I am not prepared to say whether it compares favorably with its predecessors or otherwise; but, judging by what I heard among the breeders, it was certainly up to, if not superior, to the average of the past six years.

The show of breeds, other than short-horns, at the Royal was not large. The Herefords were reasonably good, the cows very fat and very "patchy," as a rule. There was a fair show of Welsh cattle—vigorous looking animals—uniformly black and possessing a good deal of "style"; they impressed me as being really much better beef cattle than I had been led to believe from what I had previously heard of them. There were a few very good Aberdeen-Angus and Galloways, but these breeds were not largly represented.

From my stand-point the very best beef animals I saw at York were of the Devon breed. Of course they were not so large as some of the other breeds, but they were as square and blocky almost as model Berkshire pigs, on very short legs, with fine bone, neat heads, and such handlers! As an old butcher remarked in my hearing, they were "all good beef from 'orns to 'ocks."

I also liked the red polled cattle of Norfolk and Suffolk quite well. They are much like the Devons in shape and color, but are hornless. While they possess all the characteristics necessary to entitle them to rank as a distinct breed, yet it is conceded that the cattle of Suffolk have been a trifle coarser than those of Norfolk—a difference, however, that is fast disappearing under the system of blood-mingling that is now practiced between the two counties. In addition to a high order of merit as beef producers, the Norfolk and Suffolk breeders claim great superiority for the cows of the breed as milkers, and certainly their published records go far toward sustaining the claim. To those who have a decided preference for hornless cattle, yet with a dislike for the black color, I can heartily recommend these beautiful red cattle of Norfolk and Suffolk.

The show of Jerseys at the "Royal" was much larger than that of any other breed, except the short-horns, and many of the cows carried with them evidences of large milking capacity. The Ayrshires were not conspicuous, either in numbers or in quality, and the honors in the dairy classes were carried off by the short-horn cows. I may remark here that the prevailing type of short-horn cow, as exhibited at the "Royal," approaches much more nearly to that of the model dairy cow than does the type usually met with in our American show-yards. They are more after the "wedge-shape" pattern—heavy in the hind quarters and tapering toward the head—than is considered the highest model of form in a beef-producing animal.

Next to the "Royal," the most important agricultural show of Great Britain is that of the Highland Society, held this year at Inverness. The leading feature was, of course, cattle, and of these the Galloways, Highlanders, and Aberdeen-Angus took the lead. Many who had made entries here, however, as at York, were deterred from bringing their cattle on account of danger from the wide-spread infection of foot-and-mouth disease. Among others there were many entries from Sir George McPherson Grant's famous Ballindalloch herd of Aberdeen-Angus cattle, but fear lest they might incur the dreaded infection finally led Sir George to keep his favorites at home, although several of his choicest animals had been especially fitted for this show, and with every prospect of carrying a large share of the honors back with them to Castle Ballindalloch.

The short-horns were not numerous, but there were a few good ones; notably Goldfinder, a yearling bull bred by William Handley, Greenhead Milnthorpe, got by Sir Arthur Ingram (32490) out of Princess Flora, by Alfred the Great (36121). This young fellow, a beautiful red roan, exhibited by James Bruce, who is recognized as one of the best feeders in all Scotland, was shown in the very pink of condition, and is certainly the best young bull I have seen this year. He deservedly won the highest honors in his class and also the gold medal as the best short-horn bull of any age in the show. Among the other short-horns there was nothing particularly noticeable; and certainly any of the American herds mentioned above would have had an easy victory over the best of them in the show ring. I cannot help repeating here my strong conviction that our best American short-horns are superior, as a general thing, to the best I have seen here, either in the shows or at home on the farms of the breeders. The best specimens of the breed I have seen (with the exception of the young bull above mentioned) were on the pastures of the farmers of Aberdeenshire—beautiful, smooth, fine-boned cattle—such as would delight the heart of a Smithfield butcher.

The West Highlanders were numerically stronger than any other breed at Inverness, and with the Scotch people they appear to be prime favorites. I fear, however, they are too slow in maturing, and too small to be of value to American breeders. Hardiness they undoubtedly possess to an unusual degree, but if hardiness alone is what our Western ranchmen want they can get plenty of that characteristic by taking a cross back to the Texan or to that still more hardy and better "wrestler," the American buffalo. I take it that what is wanted by our Western ranchmen is a cross that will give earlier maturity, fineness of bone, lightness of offal, and a greater tendency to take on flesh. The quality of hardiness is already attained in the foundation stock.

For the same reason I am rather disposed to question the desirability of the Galloway as a cross for our Western ranchmen. Hardy they undoubtedly are, and of most admirable form in carcass, but I have a fear that this cross upon the foundation stock in use on our Western plains will be found coarse-boned and slow in reaching maturity. I venture this as an opinion, based entirely upon the prevailing type of these cattle as I have seen them in their native country. When mature I am inclined to think they are superior in shape of carcass, judged from a beef-producing standpoint, to their rivals, the finer-boned, finer-haired, and earlier maturing Aberdeen-Angus, but as a cross for the purposes above indicated I certainly look for much more satisfactory results from the latter breed, unless it be upon herds that have already been improved by several crosses with the earlier maturing breeds.

Among the herds visited by me in this country I have been especially pleased with the short-horns of Hugh Aylmer and Amos Cruickshank, and the polled herds of Sir George McPherson Grant and Mr. George Wilken. Mr. Aylmer is an ardent admirer of the Booth sort, and as his breeding is confined to that line his herd may be fairly taken as a representative one of that family. His cattle are distinguished for blocky, beefy form, on short legs, with well-sprung ribs, and straight top and bottom lines. Not remarkable for fineness or smoothness, or style, or finish, but certainly most excellent beef-producing cattle.

Mr. Cruickshank is not a follower of either Booth or Bates. He has been breeding short-horns at Sittyton for about fifty years; says it has always been his aim to breed good short-horns, regardless of the caprices of fashion; has tried bulls of both Bates and Booth sorts, but thought they did his herd harm rather than good, and so discarded first one and then the other. The bull that, in his opinion, has been most productive of good results in his herd—the greatest short-horn bull, in his judgment, that has existed within the past fifty years—was Champion of England, a bull bred by himself. He used this bull on his herd about ten years. Among the sons of the old bull he regards Roan Gauntlet as the best—but little, if any, inferior as a getter to his illustrious sire. He has now about 120 females in his herd, nearly half of the number being red, the balance red-and-white and roan, with a few all white. His cattle were not in high condition when I saw them; they were running out on pasture, and had not been in stable, nor had they seen any other feed than grass since last spring. I was particularly impressed with the evidences of early maturity in this herd; indeed I have nowhere else seen such perfectly developed *cows*, among yearlings and two-year-olds, as at Sittyton; and it is here I think that the chief excellence of Mr. Cruickshank's breeding lies. This feature has given character to the short-horns of Aberdeenshire, and so universally is it recognized, that even the breeders of the Aberdeen-Angus cattle of that region all concede, so far as I heard an expression of opinion, that up to two years old no breed will show such a gain as the short-horn. Mr. C. says his aim has been to produce a first-class beef animal at the earliest possible age; and in selecting his breeding bulls, he says: "The only questions have been, is he a good short-horn? and, Will he nick well with my herd?"

I have seen much of the Angus-Aberdeen cattle on their native heath—on the pastures of the common farmers of Aberdeenshire—and I certainly like them well. They are good-looking, smooth, fine-boned, early-maturing cattle wherever you see them. The Ericas (a family of this breed), as bred by Sir George McPherson Grant, pleased me best, on account of their comparative lightness of bone and compact, level, beefy, carcasses. Sir George has either succeeded in fixing a strong family type in these Ericas, or else he has shrewdly retained those that approximate closely to this type among those bred by him, and has sold the rest. The Prides (another popular Aberdeen-Angus family) are larger than the Ericas, and certainly have not quite the finish and style of the latter, but, judged by the show-yard triumphs of this year, they are not a whit behind them in popular favor; indeed I am inclined to the opinion that the show-yard ledger of this year displays a decided balance to the credit of the Prides. But this may be, to a considerable degree, owing to the absence of the Ballindalloch herd from the Highland show.

I regret exceedingly that I was compelled to forego the pleasure of a visit to Herefordshire. The "white-faces," as the Hereford cattle are often called, have gained so firm a footing and have become so widely popular in America that I very much desired to see them on their native pastures. I had arranged, while at the Royal show, to spend all of the last week of my stay in England among the Hereford breeders, timing my visit there, by special invitation, with reference to an important gathering of Hereford breeders that was to take place at that time, but at the last moment I was compelled by urgent business demands to send my regrets to Hereford and turn myself homeward, without the coveted visit to Herefordshire, as well as to many other places that I had hoped to see before my return. What I have seen of this very popular breed, however, has only confirmed my previous favorable impression of them as grazing cattle. But must I be permitted to say of them, as I have already said of the short-horns, that judging by what I have seen of them in both countries, the enterprise and good judgment of American buyers have already transferred the cream of the breed to the United States.

ENGLISH AND SCOTCH DRAFT HORSES.

Another very prominent feature of the English and Scotch show-yards was the cart or draft horses, the Clydesdales taking the lead in Scotland and the shire horses in England. So much crossing and mingling of blood has been practiced by the horse breeders of these two countries that it is really very difficult to tell where the one leaves off and the other begins. In fact, I have never yet been able to find a horseman whose eye was critical enough to enable him to say positively whether a given animal belonged to the one or the other breed, unless he knew beforehand who the animal belonged to, or where it came from. But as each breed now has its own stud-book it is not unlikely that a greater distinction than is now apparent may grow up in the near future. Judging by what appeared to be the prevailing type of the two breeds, as I saw them, I should say, if there is any difference in them, that the shire horse was relatively better and stronger in the heart, back, and flank, than his Scotch rival; but, as before remarked, the most critical horseman would be puzzled to give a good reason why they might not be classed as one and the same breed. They are certainly most excellent horses, and for heavy draft it is doubtful if their superiors can be found in any country. Large numbers of them are now annually being taken to the United States, the honors being about equally divided between them and the Percheron horses of France. The Clydesdale and the Shire horses have each had a stud-book for some 5 or 6 years past, and the effect has doubtless been to cause greater attention to be paid to genealogy than was possible under the former order of things; and American importers now have a much more reliable guaranty of purity of blood than they could possibly have without these published records. With the publication of the Percheron stud-book, to which I have before alluded, it may now be said that our American importers have a stud or herd book to rely upon for every breed of horses and cattle that is brought to our country, whether from Great Britain or the Continent.

DANGERS FROM INFECTIOUS DISEASES.—SUGGESTIONS AFFECTING OUR EXPORTS OF LIVE CATTLE.

In conclusion permit me to say that I am aware of the fact that I have only imperfectly covered the subjects to which my attention was directed by you in my appointment. The time which I was able to give to each branch of the subject was necessarily brief; and some of them I have been compelled to forego almost entirely. I cannot close, however, without again calling attention to the very general prevalence of foot-and-mouth disease among the cattle of Great Britain, and the great danger which exists of the transportation of this pest to our own herds and flocks, through the frequent importations that are now being made from that country. The system of quarantine now being maintained by our Government against all imported cattle affords very good security against infection, through the bovine species; but sheep, swine, and goats, are equally susceptible to this disease, and are quite as likely to bring it to our shores as are the cattle. Unfortunately there is no law authorizing a quarantine of any imported animals other than cattle, and herein lies our greatest present danger. It is to be hoped that Congress may be induced at a very early period in its next session, to confer the necessary authority for enforcing quarantine against sheep, goats, and swine, as well as cattle from all foreign countries, so that our flocks and herds may be effectually guarded against this the most infectious of all animal plagues.

I deem it important also to call attention to the fact that the establishment of a rigid system of inspection of all live cattle exported from this country and a strict supervision of the ships used for the purpose of carrying the cattle from our ports, to the end that none but healthy cattle shall be placed on board and that the facilities for promoting the health and comfort of the animals during the voyage shall be ample, is of the highest possible importance in its bearing on the future of our export trade with Great Britain. In a conversation between Professor Brown, the chief veterinary adviser of the British privy council, and myself upon this point, he dwelt with especial emphasis upon the importance of attention to these matters on the part of our Government. Americans as a class are very jealous of governmental interference with trade and commerce, and cattle shippers especially are opposed to any regulations that shall interfere in the least with their present freedom of action. But the interests of the farmers of the United States, the men who raise the cattle for export, are paramount to those of the few who are engaged in the export trade. And no consideration of a very slight reduction in the per cent. of profit which these dealers may realize should be allowed to stand in the way of the adoption of regulations which are absolutely essential to the permanence of the traffic.

Respectfully submitted.

J. H. SANDERS.

CHICAGO, *October* 30, 1883.

REPORT OF DR. RUSH SHIPPEN HUIDEKOPER.

SIR: Twenty years ago an International Cattle Show was held at Hamburg, Germany. This exhibition attracted a great deal of interest; it proved a financial success to the citizens who guaranteed the cost; it brought valuable agricultural products and animals from England France, and other countries, and it was encouraged by numerous exhibits and an active committee from the United States. The effects of this exhibition in North Germany were lasting. Valuable breeding animals were bought from the foreign exhibitors, and the numerous German farmers who attended saw and appreciated the improved methods of tilling the soil, breeding, caring for and feeding their animals, and economizing the products. Some of the committee of the exhibition of 1863 were still taking an active interest in agricultural matters when the present Hamburg Cattle Show was proposed, and they found a prompt support in offering to the people another "concours" to show the advance which had been made in the two decades. In the exhibition of 1883 but few of the foreign countries lent the support which such an undertaking deserved, and the exhibits were confined almost entirely to Germany, with her closely related neighbors of Holland, Denmark, and the Austro-Hungarian Empire.

The exhibition was opened on the 3d of July, 1883. The buildings were well arranged and every accommodation for the animals was provided, the details of which will be found in the accompanying catalogue.

The exhibits were divided into nine sections, viz:

1. Horses.
2. Horned cattle.
3. Sheep.
4. Swine.
5. Bees and the apparatus connected with their care.
6. Fish.
7. Fowl.
8. Stalls, buildings, agricultural implements, &c.
9. Scientific apparatus and methods of teaching all matters pertaining to agriculture.

SECTION I.—HORSES.

Division A.—Classes 1 to 4 were composed of thoroughbreds, which presented no particular interest. Among the stallions one of the best was an American, "Imagination," born 1874, by Longfellow out of a Lexington mare.

Classes 5 to 6 contained several excellent Arabs, which have been of great use in Germany, as in France and elsewhere, in improving the local breeds of horses before crossing them with the thoroughbreds.

Division B.—Classes 7 to 12 called for riding, hunting, and cavalry horses and mares, of three years and over, raised in Germany, Austro-Hungary, Denmark, Sweden, and Norway. These classes were largely filled with good animals, especially from Austria, as the horses from this country showed much more blood.

Division D.—Classes 13–15 were for the same class of animals from any country. England only of foreign countries was represented with several good animals from the Stand Stud Company, but the first prize was awarded to the representative of the Royal Prussian stud. In this division American horses could have been shown with special profit. Equally good horses might have been sent from New York State and the whole Alleghany ridge, with an advantage of price on their side, even with the cost of transportation and risk added.

Division E.—Foals of the previous classes.

Division F.—Heavy carriage horses.—This division was complete in stallions, mares and colts, and presented, perhaps, one of the richest shows among the horses. From the many studs represented the most useful for the United States would probably be the animals of the Grand Duke of Oldenburg. These are magnificent beasts of full 16 hands, short coupled, fine, strong neck, legs well placed (*d'aplomb*), and good feet. The developing horses are of sufficient size for the farmer to use economically while he is preparing animals, which, if they are at fault as valuable carriage horses, are still strong enough to sell or keep for hard work.

Division G contained, in addition to the preceding, a few good English horses.

Division H.—Strong horses for agricultural and industrial work. The Clydesdales were entirely wanting. Several fine representatives of the Shire horse were shown, the largest weighing some 1,900 pounds, but all except a stylish mare were sleepy and had unfortunately flat feet.

Classes 31 to 34 failed to obtain any representation from the superb French, Belgium, and Norische or Pinzgauer races. The first two are so well known in the United States that it is useless to speak of them, but the Pinzgauer should be better known. Originating (historically) in the Pinzgauer and Styria these, immense active horses have ever since been kept in all their purity. They were described and drawn from by the ancient Romans, and in the Middle Ages they furnished the mounts of most of the German and North Italian warriors. Their full blood has always exercised a strong influence upon the countries directly surrounding the South of Austria, but the limited territory in which they are produced in their purity has been a barrier to their wider dissemination, and of late years their export has been almost entirely limited to Munich and Vienna. Fine examples of this race can be met with in these two cities, but they are best seen on the Styrian Mountains, where they usually work with a load on the back and go over the heights, on a narrow foot path, as surely as a mule, with the advantage of greater speed and the strength for a greater load. As our agriculturists are now bringing so many of the large European horses to America, and the price of English and French horses have advanced so much in the last few years, it would be well for them to pay attention to these animals, which can be procured for less money. From Denmark were shown some fine horses with a good deal of blood and weighing from 1,300 to 1,500 pounds.

A few Ardenners were exhibited in harness, but none of the pure type which was so renowned in the old posting service, and has now become so rare, as it has unfortunately been rendered coarse and lymphatic by crossing with the large Flemish and English animals.

SECTION B.—PONIES.

Among the ponies no type or race was especially exemplified, except from Norway. The Scandinavian horses are magnificent heavy ponies of 13 to 14 hands, with short backs well modeled, short, strong set legs, good feet, and an intelligent head with a large open eye.

These animals keep in good condition with a small quantity of poor food, and are adapted to mountain work. They walk very fast and trot wonderfully. They are good workers in harness, though they are usually used under saddle, and carry the big Norwegian peasant as easily as they would a child. They could be profitably raised in any ot the mountainous districts of America, and would make an excellent mount for the cavalry in the West. Their price is very low, and the cost of transporting them would be less than for large horses. There was no exhibit of mules and asses.

SECTION II.—CATTLE.

The show of cattle was the great feature of the exhibition, although it was almost confined to the series of coast cattle. The present division of these cattle deserves special attention, as the American nomenclature has been decidedly arbitrary.

It is only within recent date that the proprietors and agriculturists have become convinced of the necessity of establishing definite lines between the cattle of each locality, where the animals showed some particular merit; and in order to retain the purity of their herds, which was threatened by the increased facilities of commerce, they have found the solution of their problem in the establishment of "herd books."

The catalogue called for a first division into the Marschschlage, or those families of animals coming from the lower country and into the Geestschlage, or those coming from the higher and more inland countries. In the Marschschlage series are the East Frisians, Oldenburgers, Hollanders, Wilstermarsch, and Breitenburgers; in the second series there are families from the first three of the preceding, the Angelers and the Jutlanders. Of these groups the Wilstermarsch, Breitenburgers, Angelers, and Jutlanders are in general terms "Holstein" cattle, and, except the last, have nothing whatever in common with the West Frisian, Oldenburgers, and Hollanders, which are the so-called Holstein cattle in America. The generic name of "Holstein" as applied to any race of cattle does not exist in North Germany or the Netherlands. The Wilstermarsch and Breitenburgers present no special interest for the American, as they are simply two good breeds of cattle which are fair milkers, and owe their milk qualities to the crossing of short-horns on the large native race, a bony and coarser cattle. The Jutlanders, Oldenburgers, and a part of the Frisians, especially that part of the latter to the east, have also felt the influence of the short-horns to a very great extent, but the native breeds were generally strong enough to retain their color and their milking qualities. The continental preference, which formerly bought only white short-horns, aided probably in the retention of the white and black color. While some of the families of those breeds are almost typical short-horns in form, a large number approach the Dutch and Frisian so closely that mistakes between them would be excusable. However, the lower flank, the larger hip, the finer development of the thorax, the shoulder-gutter, the fine head, and the other milk characteristics distinguish the families from Holland and Friesland. At present the most of these cattle, if of any value, are registered in the herd books. While the name of a herd does not affect its value, it would be better that the proper name should be given in the establishment of foreign herds in the United States. The two groups of these cattle, with their predominating qualities of milk or beef, should be better understood and separated at the outset.

The Angeler cattle are a beautiful race of milkers, with most of the characteristics of the Channel Island cattle, including the richness of

The Austrian and Russian cattle were wanting. A few Rhenish (Jura) and Scandinavian cattle presented only a zoötechnical interest, and the great milk race of Flanders and the North of France, and the Salers and other great work and beef cattle, were entirely absent. The short-horns and Ayrshires were shown both from German and English herds that would readily have yielded the prizes to American representatives had these been sent. A test of the quantitative and qualitative milk production of the various races was carried on during the exhibition, of which an analysis is subjoined.

SECTION III.—SHEEP.

This section was only notable for the magnificent specimens of the Rambouillet merinos, which are owned in quantities in many parts of Germany.

SECTION IV.—SWINE.

The exposition of hogs contained many fine Yorkshires and Berkshires, both from German and from English proprietors; but the finest show was in Poland Chinas, which were almost all imported from the State of Ohio, and they attracted a great deal of attention and favorable comment.

SECTION V.—BEES.

In this division was a complete collection of the breeds of bees, with all the apparatus connected with their raising and the utilization of the honey and wax.

SECTION VI.—FISH AND FISH BREEDING.

Besides a show of the implements connected with fishing, a few models of fish-breeding and artificially developed fish were shown, among which was the fine collection of carp from the farms of Adolph Gaschat Kaniow, in Galizia. This exhibit requires, however, no details after the larger ones which have recently been held.

SECTION VII.—FOWLS.

As no foreign exhibit was made, the collection of fowls can best be criticised as being composed of German birds. The representation of pigeons was wonderful, and the catalogue will probably be of interest to breeders of these semi-luxurious but useful birds.

SECTION VIII.—STABLING, MACHINES, AND IMPLEMENTS.

The moderate exhibit of wagons, agricultural implements, &c., was almost entirely from the hands of German and English workmen, and certainly would have been much more valuable if it had had the addition of the standard American machines. The few machines which were modeled from American patents were of old patterns.

SECTION IX.—SCIENTIFIC INVESTIGATION AND PRODUCTS IN REFERENCE TO ANIMAL INDUSTRY.

This section contained an exhibit which would possibly have been one of the least understood at first sight by many Americans, but when once studied would have been thoroughly appreciated and would have proved of great usefulness in showing the extent to which European governments interest themselves in aiding the agriculturist, by furnishing him with properly educated men to assist him in the selection and care of his land and animals; and to care for his animals and protect them when they are sick or are threatened with the epizooties. The first collection was a well selected museum of the books, specimens, apparatus, and zootechnic products from the Royal Prussian Agricultural High School in Berlin.

The second was a similar collection from the Saxon Agricultural School. This included a complete display of wool from all the ovine races and breeds, with an appendix giving a detailed report of the feeding and producing cost of the animal which furnished the samples.

From the veterinary department the exhibits contained a collection of skulls of all the domestic animals, anatomical and pathological specimens, injected preparations, parasites and wax models of the same, apparatus connected with the use and care of the domestic animal, &c. An interesting selection from the library showed the gradual development of literature pertaining to agriculture.

The veterinary school and school for blacksmiths, of Saxony, were also represented by complete examples of their methods of teaching and the means employed in their practical demonstrations.

Instrument makers showed microscopes, trichina microscopes, thermometers designed for stable and dairy use, lactometers, apparatus for qualitative milk analysis, &c.

Agricultural and veterinary literature was completely represented, and contained many useful models of records for dairy and farm use, and an extensive collection of all existing stud and herd books. Silk and bee culture was shown by a full series of the natural insects and their products, and a duplicate series of the same, enlarged in wax and papier maché. There were also competitive prizes for essays and designs for stables and farm buildings, to be adapted to flat or hilly countries; essays on the hygienic and other arrangements of farm buildings, manure, &c.; on the removal of animals from burning buildings; on animal production (breeding), &c.

Throughout the entire exhibition the most rigid measures were observed in regard to the sanitary police. A sufficient corps of veterinarians was constantly on duty, under the direction of Professor Johne. All animals before being admitted to the show-grounds were passed through an inspection yard and carefully examined. A constant watch was continued, and notwithstanding the immense number of animals collected from all of Central Europe and transported through the great centers of animal commerce on railway wagons and by other means of public conveyance no cases of contagious disease were detected, and none developed during the ten days on the grounds. This fortunate result is justly to be attributed to the present complete and thorough precautions taken by the German Government for the protection of its animals. Each district is provided with an official veterinary surgeon, to whom all cases of contagious disease must be reported, and this official is empowered with the proper authority for enforcing the law. Each department has a superior veterinarian, to whom the district veterinarians report, and he is invested with greater powers. If the proprietors make an immediate report of any suspected case of contagious disease on their farms, they are liberally indemnified in case of loss, after the official investigation, which is one of the official acts in Germany, and executed promptly. If, however, a proprietor attempts to conceal any case of contagious disease in his animals, he not only forfeits any right to indemnity, but is punished. A veterinary surgeon who attends such cases and does not report them is even more severely punished. This law of liberal indemnity and punishment combined has proven most satisfactory; it leads to the prompt declaration of any outbreak of disease, and allows the Government to exert active measures at the outset. In addition to these rigid measures, the precautions taken on all means of transport for their proper disinfection are thorough, not only in the law but in practice. Railway cars which have served for the transport of any of the domestic animals are immediately disinfected. The large railroad yards are provided with special tracks at the side of a building containing boilers for an unlimited supply of hot water. After the car is swept it is washed from a hose with water as hot as can be handled; after another sweeping the interior of the car is washed with water heated to 70° C., with soda added. This most satisfactory means of disinfection is carried out at an expense of only two marks (50 cents) a car, and is paid by adding that amount to the freight bill.

The slaughter of all contaminated animals, whether executed as a measure of sanitary police or for food, is done under the supervision of a responsible veterinary inspector, and so all attempts to evade the law for a little additional gain are avoided.

It is greatly to be regretted that Americans could not have been enabled to exhibit their agricultural products, especially animals, at Hamburg, that they might have shown the great resources of the United States, and, what is more important, the facility with which these resources can be placed at the disposal of the European market, as this is as yet little appreciated outside of England. The cavalry horse and the beef cattle are now the two articles which offer a favorable and profitable field for export, but it will be but a very few years before the heavy draft horse can be added to the list. For the establishment of an active and paying commerce, however, the European Governments will demand a greater security and guarantee of the health of the animals shipped to them than now exists. The necessary encouragement for the education of a sufficient number of men to carry out the State laws and the work of the Government in regard to the diseases of our animals will aid very materially not only in our own protection, but in the advancement of our cattle trade with Europe.

Very respectfully,

RUSH SHIPPEN HUIDEKOPER, M. D.,
Honorary Commissioner.

Hon. GEORGE B. LORING,
Commissioner of Agriculture.

OUTBREAK OF SOUTHERN CATTLE FEVER IN KANSAS.

REPORT OF M. R. TRUMBOWER, V. S.

Hon. GEORGE B. LORING,
Commissioner of Agriculture:

SIR: In obedience to your telegram, dated at Washington, October 9, 1883, requesting me to proceed at once to Harper, Kans., there to investigate an outbreak of disease among cattle, I forthwith made the necessary preparations and left on the 4 o'clock train the same day, arriving at Harper on the morning of the 12th. My instructions not having yet arrived, I remained in town and made inquiries of different individuals in relation to the cattle disease. I soon became overwhelmed with reports of the magnitude of the outbreak and extent of losses throughout Harper and Barbour Counties. I received many reports from different individuals, stating that such and such persons had cattle dying daily at that date. After deliberating over the matter, I came to the conclusion that I could do no better than to remain in town over Saturday and have some person who was well acquainted with the people to point out to me the men who were then losing cattle, as many of them would probably be in town on a Saturday. Here I met with disappointment. I met a number of men of whom it was said that they were losing cattle daily, but on making a direct inquiry they almost invariably said: "No; *they* were not losing any now," but named some other person who was. I soon found out that the better plan would be to go directly into those sections where the greatest mortality had prevailed, and there endeavor to find suitable subjects for examination. Therefore, on Sunday morning, the 14th, I made arrangements with Martin Cochran, of Harper, who is well acquainted with that country, to take me out into Barbour County. We left Harper about 8 a. m. The first place we stopped was at Dr. Joseph Brockway's, on the Nine Cottonwoods Creek, 18 miles southwest of Harper, in Harper County. He made the following statement: That he moved his cattle, numbering 75 head, from his place of residence, taking them northwest 2 miles, on or about the 1st day of May; that on or about the 10th day of July his cattle began to die, and 10 head died within eighteen day's time, nearly all of them being two and three year steers. The disease then abated, rainy, cloudy, and cool weather supervening. On the 1st of August 4 or 5 were still sick, but making a slow recovery. On the 12th of September the disease broke out again, and 13 head more died in rapid succession. He then fenced in a pasture for his cattle, placed them therein, and has not lost any since.

The Boyd herd of cattle (suspected—of which more hereafter) were driven on their way west from Harper into Barbour County, along a freight road which crosses his range, upon which road also oxen attached to wagons and small herds of cattle were driven back and forth all summer, and cattle passed in the care of herders every morning.

Mr. E. Walden, 2 miles north of Dr. Brockway's, lost all the cattle he had—7 head. They died in the month of September. His cattle have been near the Boyd trail, and also near the Anderson trail (another suspected herd).

The next place I visited was William A. Wood's, on the line of Harper and Barbour Counties. He made the following statement:

> Mr. Boyd, in the month of April, crossed my range with his cattle; I drove my cattle back, 13 in number, to allow him to pass. A few days later a second herd, numbering 900, were driven through on the same trail. I again turned my cattle back to allow this second herd to pass; they stopped on my range to graze for two hours; they then went west to the Little Sandy, where they turned and went northwest. These cattle were long-horns. I picketed 1 cow and 2 calves on the trail of these two herds. The cow was taken sick on the 1st of October, and died on the 12th. All of my cattle crossed this trail daily; 5 of them were taken sick, but only the 1 died.

I went out on the range to see the one that died on the 12th (two days ago). I found her in a fair state of preservation, and decided to make a *post-mortem* examination. I removed the wall of the chest and abdomen of the right side. Found the lungs in a nearly normal condition; the pericardium contained four ounces of bloody colored serum; the heart was extensively ecchymosed on both the external and internal surfaces, the endocardium being somewhat softened and paler than normal; no heart clots were present. The liver weighed 13 pounds, the enlargement being due to hyperæmia; gall bladder contained 10 ounces of a dark olive colored bile, of a thicker consistency than normal; spleen weighed 4½ pounds; contents, a dark purple semi-fluid pulp; a general disintregration had taken place; urine bladder empty; kidneys normal in color and size. The fourth stomach presented extensive congestion of the mucous membrane, especially towards the cardiac end, and many small erosions exposing the vascular membrane were visible near the pyloric end; redness and congestion, accompanied more or less by thickening of the mucous membrane of the small intestines, was manifest upon close inspection; interstitial extravasations of blood between the membranes of the cæcum and also of the rectum were found, and the feces coated with mucus mixed with blood. The uterus contained a male calf six and a half months old; the liver of the fetus weighed 1½ pounds; spleen apparently normal; pericardium contained 2 ounces of very dark-colored bloody fluid, also an abnormal amount of bloody colored fluid was discovered in the thoracic cavity.

I examined a white cow, six years of age, which was supposed to be recovering; she had been sick for two weeks; pulse 112, temperature 106° F. She was lying down. I made her get up; her gait appeared very stiff and painful, and as she slowly moved away she voided bloody-colored urine. She eats and ruminates. I examined another cow which had been sick for ten days; pulse 60, temperature 104° F; very thin in flesh; eats and ruminates. October 24 saw Mr. Wood in town; he stated that both the cows which I examined were alive and gaining in strength and health.

From Mr. Wood's place we proceeded to Mr. Jesse Boyd's, in Barbour County. Here we remained over night. During the evening and the following morning I received the following information from Mr. Boyd and Mr. Cochran, viz: Mr. Martin Cochran, of Harper, spent nearly all winter at Judsonia, on Red River, White County, Arkansas. Mr. Cochran bought 342 head of cattle in this county, most of them being in a very poor condition, attributed to cold weather and insufficient feed. One hundred of this number were bought 12 miles north of Searcy. Mr. Cochran collected 242 head and drove them to Judsonia on or about the 24th day of March. Mr. Jesse Boyd came to White County, Arkansas, about the 1st day of February. He bought 228 head of cattle in this county, brought them to Judsonia, and there he and Mr. Cochran pooled their cattle, the combined herd then numbering 470 head. They drove them 55 miles to Conway, a railroad station, adding at Searcy 100 head more (those which Mr. Cochran bought 12 miles north of Searcy). They shipped at Conway, on the 1st day of April, the whole number of the combined herd (570) and unloaded at Harper, Harper County, Kansas, on the 5th. About 50 of them died on the cars before they arrived at Harper; got down in the cars and were trampled to death. At Harper these cattle (520) were kept for three or four days, ranging north of the railroad track and traveling over a space of 2 miles, the range extending northeast to Sisson's Grove and to a small creek where they had to go to water. At night they were yarded at the stock-yards.

Twenty of them being disabled were watered in the yards and fed on corn, cane, and millet-hay for a week or longer. On the 9th of April Mr. Boyd drove 500 head of them from the stock-yards, in a southwestern course, to the edge of town, passed I. J. Campbell's field, thence west 1 mile, then southwest 1 mile, west again 8 miles, crossing L. M. Pratt's range one-half mile north of his residence, then came on the road at the school-house on Bluff Creek, thence along the main road as far as Richard Botkin's place, beyond whose place they encamped one night. From there they left the road to the south of the trail, but came back to the road again at W. E. Kline's. From Kline's they passed in a southwesterly direction to the Nine Cottonwoods Creek, east of Mr. Gardner's; here they stopped the second night. From this place they passed due west until they reached Boyd's range, on the Little Sandy Creek, 4 miles across the Harper and Barbour County line. From Boyd's range 102 head of these cattle drifted away and were gathered in again in the round-up in June and July. They went south to the line of the Indian Territory, west 12 miles to Medicine River, east 2 miles to the limits of the range. On the 10th day of July several of these cattle were noticed to be sick, and in a week 8 were dead. Mr. Boyd, at the same time that he held this herd of cattle, also had 140 head of Arkansas cattle which he brought in last year, and were wintered by him; he had 20 head of natives besides, which were also wintered by him. At the time that the recently imported Arkansas cattle began to die, two of the natives also became sick and died, both the latter being yearling heifers. All of these three different lots of cattle were herded on the same range and drank out of the same creek until the latter part of July, losing 8 of the suspected cattle, two of the natives, and none of the wintered Arkansas cattle.

The 20 head of crippled cattle which Mr. Boyd left at Harper were driven into I. J. Campbell's pasture field, located at the western edge of town, on or about the 18th day of April; there they intermingled with 150 head of native cattle belonging to Mr. Campbell. They remained in Campbell's pasture for a week, then they were driven out over the trail of the previous lot of cattle, as far as L. M. Pratt's, where they remained over night grazing with some of Pratt's cattle. From Pratt's they went in a direct course to Boyd's range.

On the 26th day of July, Mr. Jesse Boyd was arrested and brought to trial for bringing into the State of Kansas, contrary to law, wild and undomesticated cattle, which had spread a disease among the natives known as Texas fever. Four hundred and thirty head of the Boyd cattle were seized and quarantined by the sheriff of Barbour County, were placed into Mr. Cook's pasture field, 2 miles south of Mr. Boyd's house, and there were kept under surveillance by the sheriff's assistants. The witnesses on part of the State were: William Garrison, Mr. Hufacker, David Clough, W. W. Cook, Albert Cook, M. B. Moore, George M. Gardner, J. H. Warren, A. B. Rannals, W. E. Mattox, all men holding cattle close to Mr. Boyd. Mr. Boyd was fined by the court in the sum of $300, and sentenced to imprisonment in the county jail for a term of one month. He took an appeal to the higher court. His second trial has not yet taken place.

On the 15th of October, I went to see Mr. Jackson, who has charge of the quarantined cattle. He reported six deaths among them since the 29th of July. On the 18th I was again at Mr. Boyd's place. I then tested the temperature of 4 head of his wintered cattle which registered as follows, 101.8°, 100°, 101.4°, and 100.9° Fahrenheit. I proceeded to the quarantined herd and tested the temperature of 20 head among them. We drove them into a branding pen. They registered as follows: One four-year old cow, 102.1°; two-year old heifer, 103.7°; three-year old steer, 101.6°; two-year old steer, 103.4°; yearling heifer, 102.6°; three-year old heifer, 103.7°; pulse, 66; two-year-old heifer, 101.8°; two-year old heifer, 102.9°; yearling heifer, 102.2°; four-year old cow with calf at her side, 102.9°; three-year-old heifer (scouring), 102.4°; three-year old steer, 102°; six-year old cow, 102.8°; two-year old steer, 102.4°; two-year old steer, 102.8°; three-year old heifer, 102.6°; three-year old steer, 102.6°; ten-year old cow, 103.1°; six-year old cow, 103.2°; two-months old calf of the latter, 104.9°; one lame cow, a stray, ox yoke brand, which has been lying around with these cattle since the July round-up, indicated a temperature of 103.7° F.

These cattle did not appear wild and undomesticated, and stood very kindly under my examination. Here I wish to mention a word about the July round-up. In the latter part of June or the first of July about 2,000 head of cattle were brought to and centered upon Boyd's range in the round-up; nearly all of the neighbors of Mr. Boyd, and many others, came to this place and cut out their own cattle. Some of the strange cattle remained several days on the Boyd range. If any cattle were in this herd that carried the germs of the Southern cattle fever, it follows that all the cattle in this round-up were equally exposed, or nearly so. In this herd of 2,000 were about 10 head of stray cattle, for which no owners appeared, and it is not known to-day where they belong, nor from where they originally came. Again, Mr. R. B. McGee, secretary of the Cedar Hills and Sand Creek Pool, gave me the names of the membership of said pool, as follows: McGee and Lovett, Jesse Boyd, M. B. Moore, William Garrison, Mr. Hufacker, Mr. Beals, David Clough, Perry Clough, John McKeever, John Elmore, Hink, Moore, Munger Brothers, J. H. Warren, E. C. Davis, Mr. Holmes, M. McGuire, and Mr. Cross. All, or nearly all of these parties, had a greater or less number of cattle in this pool during the winter or spring, where they remained until about the 1st of July. If any of the ground over which these cattle passed was capable of imparting the Southern cattle fever, or, if any cattle were in this herd carrying the germs of this disease and discharging them, then all of the cattle in the herd were exposed to such grounds or cattle. On Monday, October 15, Mr. Boyd and Mr. Cochran accompanied me to Mr. John Elmore's ranch, located on range 15 and 16, T. 33, R. 10 W., Barbour County, or four miles southwest of Boyd's range. Mr. Elmore stated that he held on his range 210 head of grown cattle and 40 calves, all wintered cattle except 38 head, which were brought in from Atchison County, Missouri, in the spring. Mr. Elmore's cattle began to die on the 1st day of July; 13 head died in two weeks; the disease then checked up but broke out again about the 1st of September. During this month 143 died, including 3 or 4 spring calves. Out of the 38 head brought from Missouri, 15 died. He states that he does not know of his cattle being exposed to any through cattle, or other diseased cattle, except the Boyd herd. Several of his cattle drifted away in the spring and were gathered in the round-up; one of them was gathered in the Territory round-up some fifty miles south of the line.

Last year Arkansas and Indian cattle were held on this range by Munger Brothers. Late in the fall Mr. Elmore brought 362 head of cattle from Northeastern Kansas and Northwestern Missouri and placed them on this range; in two weeks thereafter 6 of them died with Southern cattle fever. (See Dr. Detmer's report of last year's investigation —Moore and Elmore.) During the winter they lost 108 head, including the 6 just mentioned, out of the number.

From Elmore's we drove to Cook's and Rannal's, 4 miles southwest of the former, located on the western side of the Cedar Hills. We found no sick animals among this herd; only a small boy was with them, and he did not know how many they had lost.

I saw W. W. Cook at Medicine Lodge on the 27th, when he gave me the history of his losses. He owns a 600-acre pasture 2 miles south of Boyd's residence, which was an open range until the 12th of May, when he fenced it in with barbed wire. He bought from Mr. Perry Clough 140 head of cattle on the 1st day of April. They were wintered on this range. In the month of May 5 bulls were added, and on the 10th of June 217 head of cattle were brought from Kingman County. These 217 head were bought from three different parties—Williams, Lowry, and Fowler. He drove them in two lots. The first lot, composed of 41 head, were driven south along Sand Creek, corraled one night in Mr. Carlysle's corral, 12 miles northeast of Medicine Lodge, and then brought on the range.

The second lot, 176 head, were brought from the Chikaskia River, driven south through Dr. Wisner's range, and on south, crossing Mr. Boyd's range on their way to Cook's pasture, arriving at their point of destination on the 12th of June. His cattle began to die on the 9th of July; on the 14th, 9 were dead and 7 sick. The cattle were then moved south of Rannal's range, south and west of the Cedar Hills; the 7 sick ones were allowed to remain in the pasture, 5 of which died and 2 recovered. On the 29th of August his cattle began to die on the range, and he lost in all 75 out of 331 head; 10 or 15 that were sick recovered. Change of pasture was not beneficial in this case. Mr. Albert Cook, who took care of W. W. Cook's cattle, testified at the Boyd trial that some of their cattle got out of the pasture and others got into it, by breaking down the wires. This must have occurred between the 12th of May and the 14th of July. Mr. Rannal's, north of Cook's range, lost 30 out of 150 head during the month of September. These were close herded since the 1st of July. Mr. Rannal's and a part of Mr. Cook's cattle were exposed to the Boyd cattle, and also to all other cattle in the county round-up.

From Mr. Cook's range we went to R. B. McGee's, on the Little Salty, T. 34, R. 10 W. McGee and Lovett hold 200 head of cattle ranging on the east side of the Little Salty, 1 mile north of Hazleton. The first loss in their herd occurred on the 15th of September; 21 died to date; 8 remain sick, but are recovering. One, a 5-year-old bull, died last night or this morning. I desired to make a *post-mortem* examination of the bull, to which McGee readily gave his consent and assistance. We found him lying on the left side; a few drops of bloody water were distributed over the surface of his body. I removed the right fore leg and shoulder, the wall of the chest and abdomen. The animal was not *very* fat, but the fat was the color of yellow beeswax, with a slight greenish tint added to it. I removed the spleen, but in doing so I had to use the utmost precaution in handling it so as not to rupture its covering membrane. The organ was of an enormous size, and apparently distended o its utmost capacity, weighing after removal 13 pounds and measuring 30 inches in length. I next removed the digestive organs. The contents of the third stomach were slightly hardened; the lining of the fourth stomach presented numerous and extensive patches of denuded surface of irregular forms, and many of them having an ulcer-like appearance.

There was considerable capillary congestion of the mucous membrane throughout the intestinal canal; a few of the intestinal glands appeared to be enlarged and were changed in color to a grayish black. The liver weighed 24½ pounds; its structure was somewhat softened by a fatty degeneration, and impressions of the fingers were left upon it as is common in œdematous swellings. The gall-bladder contained 32 ounces of bile of normal color, but was rendered more or less viscid and glutinous by the admixture of mucus from the internal surface of the gall-bladder; the walls of the gall-bladder itself were thickened, caused by tumefaction of the mucous coat. The urine-bladder contained a gallon or more of a brownish-red colored urine, specific gravity 1.008. The left kidney weighed 4½ pounds; was darker in color than normal. The glandular structure of the right kidney was entirely absorbed; the pelvis and some of the larger ducts had become changed into cysts, containing a clear amber colored fluid possessing the smell of healthy urine. It presented eight of these cysts, each one distinct and separate from the other, and all taken together formed in bulk about the size of a normal kidney. After the letting out of the fluid, I had nothing in my hand except a mass of fibrous tissue—*there existed not the least remains of glandular tissue.* The heart weighed 8 pounds; was enlarged and flabby; the external surface was extensively ecchymosed along the anterior and posterior ventricular furrows; internally, the fleshy columns were almost of a black color from ecchymoses; no heart clots in the cavities. The animal seemed almost bloodless, the veins and arteries being empty and collapsed. Mr. McGee stated that this animal had been lame for the past fifteen months in his right hind leg, and that at times he thought he could not well serve a cow on account of weakness or pain in the back. In the latter part of June, McGee saw 250 head of cattle crossing his range. They were said to be going into Arizona; they looked like Arkansas cattle.

From McGee's we drove to the place of Robert J. Evans, having heard that he had several sick cattle. He lives about 6 miles east of Kiowa, Barbour County. When we arrived at his place no one was at home, but we found a dead cow lying not over ten rods away from the house; she had the appearance of having died within a few hours. I proposed to make a *post mortem* examination, even if the owner was absent. We found her resting upon the sternum, the head thrown back to the right side. She was propped up in this way by the nose touching the ground. Cadaveric rigidity had already set in; drops of blood were dried on the hair along the sides of the neck and flanks.

We turned her over on the side and I opened her in the usual way. I found the lower parts of the posterior portion of the lungs in a state of red hepatization, and considerable serous effusion had collected in the thoracic cavity. The endocardium was heavily congested, thickened and discolored; the fleshy pillars in the ventricles were of a dark purple color, and the auricular appendages were extensively ecchymosed.

Weight of liver, 20 pounds. Weight of spleen, 4 pounds. The gall-bladder contained one quart of very dark green bile, which was of a granular appearance.

A serous and bloody infiltration was discovered surrounding the kidneys. The urine bladder contained a very dark blood-colored urine; specific gravity 1.012; the organ was thickened by capillary congestion of the mucous coat, and a few ecchymosed spots presented themselves. The contents of the third stomach were very much drier than normal, and the folds presented a congested and irritated appearance; fourth stomach congested, and numerous small erosions and granular elevations were to be seen on the mucous surface. This animal did not present much biliary discoloration of the fat, and the blood was not as thin and watery as is usual in Southern cattle fever. The uterus contained a fetus about four or five months old; it presented a liver enlarged to three times the normal size; the kidneys intensely congested, and partially disorganized by softening and disintegration; spleen nearly normal in color and size; the pericardial, thoracic, and abdominal cavities contained considerable bloody colored effusions of serum. When I was nearly done with the dissection, Mrs. Evans returned, and she told me that the cow had been sick three days. Another cow was sick in the herd; after some lively running we succeeded in lassoing her; she had been sick four days; temperature, 104.5°; pulse, 96; nose dry; ropy saliva flowing from the mouth. October 20th met Mr. Evans, who reported two additional deaths, and two more sick. He brought 116 head of good grade cattle from Allen County, Kansas, arriving on his present range with them on the 11th of May. Since that time the cattle have not strayed in any direction 3 miles distant from a central point on the range. The first loss occurred on the 3rd of October, and 6 have died to date, the 20th.

We next visited Mr. Parsons. He lost some cattle last year, attributed to the bringing in on his range of Indian or Choctaw cattle by Morton and Tolliver. This year he lost 3 out of 80; they died in August. October 20th he reports 2 more deaths October 23d reports 2 head of the oil-brand cattle, on the same range with his, as being sick. Hink Moore lost 5 out of 700 head. Boyd's cattle ran with his for several weeks before the round-up in July.

We went north from Parson's to Thomas Brakey's ranch. He lives on the Medicine River, in Barbour County, 32 miles southwest from Harper and 3 miles from Kiowa. We remained with him over night.

He came from Chase County, Kansas, last September, with 300 head of fine high grade cattle. In January they were in good condition and thrived well. After a week's sleet and rain in February they began to sicken and die. During the months of February and March he lost 110 head. Some of them died in good condition and most of them retained a good appetite until death. The majority would first show lameness on the left foreleg, would then persist in lying down, and many of them lingered along from two to four weeks before they died. He fed all the corn, sorghum, and millet-hay they could eat. Two of them died in the month of May while on grass. On opening them, after death, the fat presented a very yellow appearance. Mr. Rider also had 400 head of cattle on this same range; he lost 75; they were affected similar to Brakey's.

Mr. Brakey gathered 200 of his cattle in the round-ups in June and July. About the middle of September one cow was taken sick; she died in a few days. Then others sickened; some of them lived five or six days, and others would be found sick in the morning and dead at noon.

He took them off the range and turned them into a cane field, but they still continued to die. He lost 15 head.

October 16, visited Harry Matthews, adjoining Mr. Brakey on the north; he has held 36 head of cattle in his fenced range for two years. On the 6th instant 110 head owned by Charles Blackstone, of the Eagle Chief Pool, were turned into Mr. Mathews' field. A few days ago a three-year old steer of Blackstone's was found sick; he died last night.

Post mortem: weight of spleen, 8 pounds; liver, 18½ pounds; heart, 5½ pounds; extensive extravasations of blood into the walls of the right ventricle; bile thick and grumous; urine bladder contained four pints of bloody-colored urine; fat the color of yellow bees-wax; third stomach slightly impacted with dry food.

A three-year old white steer was pointed out to me, which showed evidences of sickness, manifest by segregation, a weak, staggering gait, drooping head, and feces covered with mucus. One of the boys lassoed him. Temperature, 106°; pulse, 90. October 23, 2 more reported sick and the white one dead. I saw Mr. Ewell, secretary of the Eagle Chief Pool, in Harper on the 25th. He stated that no cattle died in their herd this season. Mr. N. Sherlock, of the same pool, corroborated the statement of Mr. Ewell.

From Mathews we drove through a drizzling rain southeast to the line of the Indian Territory; stopped at W. E. Campbell's cow camp. Campbell's pasture is fenced in, located on the Indian Strip, 3 miles wide and 12½ long. The cow-boys told me they lost 6 or 7 out of 3,500 head. They did not show any particular anxiety to be interviewed. We then proceeded to Pryor and Miller's ranch. They own a fenced range in the territory along the line of Harper and Barbour Counties. The cowboys here reported a loss of 10 out of 500 head of cattle bought from Hink Moore on the 10th of September. Last year they lost on this range 50 or 60 out of 4,000 with Texas fever. Lost last winter 5 per cent. Hold this year 6,000 head. Mr. Lathan, east of W. E. Campbell's, lost 16 out of 500.

We next came to H. Hale's ranch, 28 miles southwest of Harper, in Barbour County. He holds 300 head of cattle. The disease broke out among his herd about the middle of September. He herds his cattle on an open range. Lost 33 out of 300; 20 recovered.

The next place we stopped was at John Peters' camp. He is located on an open range 6 miles south of Mr. Boyd's. He lost, out of 155 head of grown cattle and 100 calves, 84 grown and 12 calves. Three of the calves were only 2 months old; the other 10 were March calves. One animal died in July; the remainder of the deaths occurred during the first fifteen days in September. After 94 had died, he left the herd and went home, stating, so I was told, that they might all go to ———, he was not going to stay any longer to see them die. After a few days he returned, and found 2 more dead; no deaths have occurred since his return to the herd. Let us suppose for a moment that, had he commenced to exhibit medical treatment at the time he left them, and only 2 deaths occurred subsequently, would he not be justified in believing that his treatment had effected the change?

The Peters cattle were mixed up with the Boyd cattle from the middle of April until the 10th of July. A stage road passes across the Peters range, and ox teams frequent this route. From the Peters ranch we drove to Mr. Boyd's and stopped for the night.

On the morning of the 17th Mr. Cochran and I left Mr. Boyd's and went west 3 miles to the residence of David Clough. He gave me the following history: His cattle began to die soon after the 4th of July.

He lost 35 head out of 300 during this month; the disease then disappeared. He sold 60 head of his cattle in August. In the early part of September the disease reappeared and he lost 30 head more out of 205. He wintered 150 head; among these the mortality was the greatest. The wintered cattle intermixed, more or less, with the Boyd cattle from the middle of April until they began to die. Two more deaths reported October 21. Mr. Clough and several of his neighbors made a number of *post-mortem* examinations, and he described the appearances of the pathological lesions of southern cattle fever very accurately. Mr. E. C. Davis, 3 miles northwest of Mr. Clough, on the Medicine Lodge stage road, and west of the Illinois colony, states that one of his cows died on the 1st day of July, and that the last death occurred on the 6th instant. The first animal that died had been running with the Boyd herd; the rest of them had been exposed to oxen that Mr. McGuire bought from Boyd in the spring. Loss 6, and 3 recoveries.

The next person we saw was Mr. William Garrison. Garrison, Beals, and Hufacker hold 600 head of cattle together, on an open range north of the Medicine Lodge stage road, and south of Dr. Wisner's range. The three parties live in the Illinois colony. All of their cattle except 80 head have been in the Sand Creek and Cedar Hill pool. They lost 30 in the month of July. On the 10th of September the disease broke out the second time; they then lost 55 head; 100 that were sick recovered. These same parties held cattle on this range for four successive years, and this is the first year that they met with any loss by disease. Mr. Garrison stated to me that he bought 80 head of vaccinated cattle from the vaccine farm at Webster Grove, Mo., all heifers, yearlings, and two-year-olds, with the exception of 12 head which were yearling steers.

He told me, in answer to my question whether or not the steers had also been vaccinated, "Yes," said he, "they told me they could not always procure a sufficient number of heifers; consequently, had to use young steers." He says he loaded them on the cars at the Old National stock-yards at Saint Louis, and landed them at Harper on the 18th of June. Mr. Garrison reports that the Anderson herd of cattle went as far west as P. B. Cole's range, which is directly north of their range. Mr. White, in the same colony, lost 2 cows and 1 ox out of 4 cows and 6 oxen. He kept them strictly under his care at home. He bought one of the cows in Harper County, and she came over the same road where the McGuire oxen had been traveling. These cattle died in the month of September. Mr. Pelton, in the colony, lost 2 out of 20 head, 1 in July and 1 in September. T. B. Stockstill, on the Medicine Lodge road, between E. C. Davis and M. B. Moore, holds 150 head of cattle.

Two died out of 5 that were sick. The first one was sick on the 6th instant and died on the 9th; the second one died a week later. These cattle were close-herded all summer, and were not in any way exposed to the Boyd cattle. He thinks his cattle took the disease through M. B. Moore's. We then visited M. B. Moore, directly west and adjoining Stockstill. He stated that his cattle began to die on the 11th or 12th of July, and 7 head died during the month. On the 15th of September another outbreak of the disease set in; they then died off rapidly for about ten days, when the death rate began to diminish. Altogether he lost 40 out of 123 head; 5 recovered and 3 are yet sick.

After death drops of blood would be seen to ooze out of the skin between the legs and under the jaw and side of neck. I examined a sick two-year old heifer which he was bringing in from the herd as I drove up. Her temperature was raised to 107.8° F.; she passed feces covered with mucus and blood. I also examined a white cow with calf at her side; had been sick for three weeks; temperature 103.2° F.

His cattle have been close-herded since the 16th day of July. Three of them were cut out of the Boyd herd at the round-up in June; none of the rest have been exposed to Boyd's or any other known diseased cattle, as far as he knows. On the 25th I saw Mr. Moore again, when he stated that the sick cattle which I had seen at his place were gradually recovering. Mr. McGuire, in the colony, lost 6 out of 60 head of cattle. Mr. McGuire bought two yoke of the Arkansas cattle from Mr. Boyd in the spring.

From Moore's we drove to B. F. Kemp's place, 4 miles south, and west of the Cedar Hill. He made the following statement: Out of 108 head of cattle, 65 got away in August and ranged over the same ground that Boyd's cattle had been grazing on; they remained from the evening until next morning; twenty days after this exposure they began to die; 10 were found sick in one day; they died in from six hours to ten days after they were taken sick; 15 died and 15 recovered. He knows of 4 that died which had been on the infected ground. One calf died, and 1 calf that was sick recovered. He brought his cattle from 10 miles east of Harper, on the 27th day of March; close-herded them all summer. Some of the Boyd cattle came over on his range in June and July. Every one found dead was lying flat on the side. When they got sick they quit eating.

From Kemp's we returned to Boyd's and remained overnight. Next morning, October 18, I took the temperature of the quarantined cattle as recorded in the early part of this report. We then went south to the ranch of Mr. W. E. Mattox. We were there told that he lost 7 out of 125 head of cattle. He brought his cattle on the range in the spring, and they were frequently among the Boyd herd in the early part of the summer. All of the deaths took place during the month of July.

From this place we turned and went north of Mr. Boyd's, 2 miles, to see Mr. James Roberts. He lost 1 cow on the 6th, after a sickness of five days; no others were taken sick. His cattle have been on the Boyd range several times during the summer. Mr. Reeder, who lives 3 miles east of Boyd's, held 27 head of cattle; 3 of them died during the month of July. Next we stopped at Mr. Crawford's, 2 miles west of Inyo post-office. They close-herded 80 head of cattle near home; lost none. They are on a road leading west toward the colony and north to Dr. Wisner's. Crawford, jr., reports having seen a herd of cattle passing between their house and that of Mr. Dougherty, half mile north, early in the season. They were going west.

We then drove to Dr. Henry Wisner's residence, 5 miles northwest of Inyo post-office, in Barbour County. Dr. Wisner was not at home, so I applied to Mrs. Wisner for information. I found Mrs. Wisner to be a regular M. D.; more than that, she took as much interest in the welfare of their stock as her husband did. She superintended a *post-mortem* examination of a bull that died about a week previous, and described the pathological lesions as follows: She found considerable enlargement and engorgement of the radicles and blood-vessels of the liver; the gall-bladder was distended, with a greenish-black bile; spleen very hyperæmic; ecchymosis found throughout the peritoneal sac; urine bladder filled with a highly bloody-colored urine—a deep wine color; kidneys unusually pale and somewhat enlarged; feces in colon hardened. Dr. Wisner bought in the month of July 120 head of the McMullen cattle on the Botkin range; a number of them died before they were taken home. He took them home in the early part of September, but did not bring them into his inclosed pasture-field among his other cattle until later; 29 of them died before they were placed in the field, and 1 after they were moved in. These cattle were close-herded and kept on poor and dry feed by Mr. Botkin; after Dr. Wisner took them home he fed them on sorghum, millet, and had good pasturage. On an open range, north of the pasture-field where the above-mentioned cattle were held, grazed 300 which were being close-herded. Mr. William Garrison, on or about the 20th of July, drove his cattle on to Dr. Wisner's open range; Dr. Wisner drove them back about the 1st of October. Several of the Garrison cattle died while they remained on this range, and were left to decompose near the pools and stream of water where the Wisner cattle had to drink. During the month of October Dr. Wisner lost 1 thoroughbred bull and 2 high-grade calves out of the 300 head. In the month of February Dr. Wisner brought from Waco, Tex., 7 car-loads of Texan cattle, unloaded them at Harper, and then drove them out to his place. These cattle intermixed with his other cattle during the summer. An ox-team, bought from Dr. Wisner's herd of Texan cattle, was employed all summer in hauling hay from Inyo, or near Inyo post-office, to Medicine Lodge. They are owned by Mr. Downing. This hauling was over a distance of from 15 to 18 miles; would probably take three days to make the round trip, and it is the custom of men driving ox-teams to stop any where along the road to feed and to allow their cattle to graze. These cattle passed by Moore's, Stockstill's, Davis', McGuire's, and others living in the colony and along the road to Medicine Lodge. I met the team about 1 mile east of Mr. Davis' house, and received from the driver (an Englishman) the facts as just stated. J. H. Warren, 5 miles northwest of Boyd's, lost 4 out of 40 head of cattle; they died in the month of September. His cattle strayed away and went on the Boyd range several times during the summer. On our way back to Harper we passed Mr. Gardner's place, near the Nine Cottonwoods Creek. Both the Boyd and the Anderson cattle crossed his range. He lost 1 out of 3 cows. Mr. Kepler, 1 mile west from L. M. Pratt, had his cattle staked on and near the Boyd trail all summer; no loss.

We next stopped at L. M. Pratt's, 12 miles west of Harper. He lost 6 cows and 1 bull. The first one was taken sick on the 4th day of July. All died within the space of ten days. He made *post-mortem* examinations of several that died; found the spleen enlarged to three times its normal size; gall-bladder greatly distended with bile; liver enlarged; bloody water in the peritoneal cavity; tallow of a saffron-yellow color; in one the urine bladder was filled with bloody urine, in another it was empty. Many of them passed bloody urine before death. Mr. Boyd passed one-half mile north of Pratt's house with his cattle on the 9th of April; a few days later brought 20 head of his cattle to Mr. Pratt's house and left them there overnight. Mr. Pratt had 9 bulls and several milch cows at home; these cattle grazed with the 20 head of the Boyd cattle, and also grazed over the trail of the Boyd herd. On the 15th of June Mr. Pratt took 8 of the bulls about 8 miles southwest to his herd, and brought back with him one cow and her calf. This was the first cow to sicken and die. The 8 bulls remained well. On the 23d of June he took to the herd a dry cow and a bull; on or about the 6th of July both these died. No strange cattle, to his knowledge, passed along the range where the cows and bulls grazed except the Boyd cattle. After I obtained the above history from Mr. Pratt I learned that a team of oxen coming from Fort Sill, near Red River, Chickasaw Nation, were driven through by Mr. Pratt's house and remained with him overnight;

this was in the month of June. From Pratt's they went west, passed Otega post-office and crossed John Peters's range in Barbour County. It was now getting dark, so we returned to Harper.

October 19, I met Mr. T. A. Barton, who lives in town. He stated that he had his cattle on the range north of the railroad track, but did not lose any. This morning we drove west 6 miles to the residence of Mr. F. P. Melvin; he lost 2 out of 4 head of cattle; one of these died on the 8th of October, and the other on the 17th. I went out into the field to ascertain the *post-mortem* conditions of the latter, but found that the dogs had anticipated me; had mutilated the body to such an extent as to render it unfit for further examination. Mrs. Melvin stated: "In the month of August I saw a herd of cattle going west, but I don't know where they came from nor where they went."

We then drove 1 mile further west to C. P. Bradford's. He stated that he lost 5 out of 6 head—2 bulls and 3 cows. Two died in October and 3 in September; 1 recovered. He said: "In skinning them I found the flesh blubbering behind the shoulders; urine bloody before death." This was all he could tell of the symptoms during life and the appearances after death. All of these cattle had been picketed close to the house, and small herds of cattle were frequently driven past his house during the summer. He stated, also, that Mr. Burr's cattle came over among his cattle while they were picketed, and that his cattle had not been within a mile of the Boyd trail at any time during the season.

From Mr. Bradford's we went to Louis Hildebrant's. He stated that Charles Martin had bought 60 or 70 head of cattle from Mr. Potter and had driven them west and southwest of Harper, passing his (Hildebrant's) place on the 6th day of August. Two weeks thereafter Hildebrant lost 3 milch cows out of a herd of 12; they were all taken sick, and 2 that recovered aborted their calves. The Martin cattle were taken to the Botkin herd.

Mr. Welker, 8 miles west of Harper, lives close to the Boyd trail; he settled on this range on the 1st of April. He picketed 6 head of oxen and 2 cows on the Boyd trail. No loss. We next saw Mr. S. H. Coyer, 1½ miles northeast of L. M. Pratt's. He took 18 cows into the Botkin herd soon after the Boyd cattle passed his place. One of them died on the Botkin range, in July; he then took them home, but they continued to die until 14 out of the 18 head were dead.

We then proceeded towards Silas M. Shafer's place, north of Attica post-office. On the way we met his brother, who told us that Silas lost 10 out of 18 head of cattle; they died in July and September. These cattle crossed the Boyd trail in the spring. On the 1st day of July they were taken into the Botkin herd, and remained there until the middle of the month. In the latter part of April, or beginning of May, a small herd of strange cattle were driven over the same trail that Boyd's cattle passed over. This trail is located a mile south of Shafer's house. Mr. Shafer has resided here for 5 years, and has never before lost any cattle. I saw Mr. Helbert, who lives 2 miles west of Pratt's, at Anthony, on the 1st of November. He stated that he had two cows which followed the Boyd herd on the 9th of April for a distance of 2 miles, and that during the summer they grazed over the Boyd trail, off and on, without any bad results following. We then turned toward Harper, and on the way stopped at Elijah Vian's place, 10 miles southwest of Harper. He lost 36 out of 100 head of cattle during the month of September. Their range was south of the Boyd trail. On the 20th we drove north of Harper to gather the history of an outbreak which had occurred in that locality. We first saw Mr. A. S. Woodward, who resides 3 miles north of Harper. He reported a loss of 14 head of cattle out of 32. These cattle, together with 29 head belonging to D. W. Fye, were herded on the same range all summer, said range being 1½ miles square. One cow belonging to Mr. Carpenter, another to Mr. Creighton, were also kept in this herd; both of them died, and 20 out of the 29 belonging to Mr. Fye died. The disease manifested itself about the 1st of September, and in the space of three weeks' time all of these deaths occurred. Eight in the herd which presented evidences of sickness recovered. They were all good grade native cattle. The history as given me by Mr. Woodward of the symptoms during life, and of the *post-mortem* appearances, leads me to the conclusion that these cattle died with southern cattle fever.

Proceeding from Mr. Woodward's place eastward, 1 mile, to the residence of John Challis, I was there informed by Mr. Challis, jr., that 12 out of 100 head of their cattle died and 6 recovered. Here, also, the disease appeared about the 1st of September. Forty head of these cattle were brought from Doniphan County on the 12th of May, and were driven from the stock-yards at Harper, 4 miles northwest, to Mr. Challis' herd. Two of the Doniphan County cattle died; the other 10 were of the domestic herd. On the 6th of June, 200 head of cattle, said to have been from Memphis, Tenn., were brought into Harper, unloaded at the stock-yards, and driven north 3 miles, where they were afterward close-herded by Mr. Scoby, the reputed owner. These cattle ranged south and east of the Challis herd and south of Woodward and Fye's herd, a public road being the dividing line between the three herds. I could get no definite history of these cattle. Mr. Cahlan, whose range was southwest of the Scoby cattle, states that 8 or 10 of the Scoby cattle died in the month of September; that the cattle were sold and driven north toward Kingman County; the owner then left Harper. I went to see one of the men who assisted Mr. Scoby in herding the cattle, but could get no satisfactory replies from him, other than that if I had money to pay for information he might tell me what I desired to know; that he was paid by Scoby for the services he rendered him. Some of these cattle were shipped to Kansas City, the remainder were taken northwest by trusty men who would not disclose their destination.

I went to see Mr. William A. Creighton, of Harper. He made the following statement: That he wintered 27 head of cattle. They were in the stock-yards at night with the Boyd cattle, and ranged over the same ground north of the railroad track with them several days. In the latter part of June they were driven out to L. M. Pratt's herd, passing along and over the trail of the Boyd cattle; one of them, a bull, was left at Pratt's for two weeks; he was then also taken to Pratt's herd; he died five days after he entered the herd. None of the rest of the 27 died, although all were equally exposed to the Boyd trail.

October 21, being Sunday, I remained in Harper. At the hotel where I was stopping I met a gentleman from Linn County, who told me that a Mr. Goss, of that county, lost 60 out of 70 head of cattle this summer with Texas fever. I met also Mr. Donahue, of Atchison, Kans., who holds cattle 4 miles east of Caldwell, Sumner County; he lost 2 out of 14 head in the month of September. These cattle he bought from Mr. Cox, who, it is said, lost a large number of cattle this season. Having heard that hog cholera existed at or near Wellington, Sumner County, and as you desired to secure virus for the use of the Department, I therefore took the night train for Wellington. I there made inquiries in regard to the truthfulness of the report, and was referred to the mayor of the city, Mr. Hamilton. He informed me that a fatal disease had prevailed among the hogs on several farms south of town, notably so on that of Mr. John Botkin. I proceeded to Mr. Botkin's place, located one mile south of town, found him at home, and obtained the following history of the disease among his hogs: Mr. Smith, a near neighbor, received some hogs last year from Missouri; soon after he got them home a disease appeared among them and many died; soon thereafter, Mr. Botkin's hogs began to die, and he lost nearly all he had. Those that remained well he sold in the fall, keeping no hogs over winter. The hog pasture of Botkin adjoins that of Mr. Smith. A large pond of surface water is inclosed in Mr. Botkin's pasture, and the surface water from Mr. Smith's pasture flows into this pond. In the month of June, this year, Botkin bought a number of sows and pigs, took them home and placed them in the same pasture that held his hogs last year. Within a month his hogs began to manifest evidences of disease. As there were none in a dying condition when I saw them, I requested the privilege of killing one for examination. Mr. Botkin willingly granted the request, and caught a 2-months-old boar pig. He presented the following symptoms: pulse, weak and compressible; temperature, 106° F.; considerable swelling across the nasal bones, posterior to the nares; a large ulcer, one inch in diameter, opposite the first molar tooth in the superior maxilla, forming a deep cavity in the tissues covering the alveola; this ulcer was of an unhealthy, foul, and sloughing character. Several small ulcers were present on the tongue, possessing well defined borders, surrounded by a darkened, brownish-red areola; a very offensive odor was emitted from the mouth. Several abscesses and ulcers of variable sizes, from that of a pea to a silver quarter of a dollar were discovered on the abdominal surface, and one large and deep ulcer immediately about the coronet of the right fore foot. (Several other hogs on the place presented similar symptoms to this one, but not so far advanced, the first and most prominent symptom being that of the swelling across the nose, accompanied by more or less snuffling.) They continued to eat, and do not lose flesh very rapidly; a few of them suffer by diarrhea, and colliquative diarrhea sets in before death takes place. (There is no special tendency to hide in the litter, nor is there any roseate blush present at any time during the progress of the disease, as there is in true hog cholera.) I severed the jugular vein and carotid artery on one side of the neck and bled it to death, then removed the wall of the chest and abdomen. I discovered a greenish yellow serum in the abdominal cavity; heart pale and flaccid; lower lobes of lungs in a partial state of hepatization, abscesses and tubercles being distributed throughout the affected parts. The stomach presented two extensive, irregular-shaped ulcers, apparently in process of healing, one of them measuring two inches in length; they were covered with a yellowish deposit of pus, mucus, and feed mixed, which was strongly adherent to the surface of the ulcers. The intestines contained a great number of long worms, *ascaris suillæ*, and the intestinal glands presented a thickened, infiltrated, tuberculotic condition. The liver was enlarged to three times the normal size, was of a light olive-yellow color, and numerous small abscesses were found within the structure of the organ; the biliary ducts were literally filled with worms of the same species as were found in the intestinal canal. The worms in the liver obstructed the flow of bile. The gall bladder was filled with bright green-colored bile, thinner and lighter in color than normal. The urine bladder contained three ounces of light green colored urine; kidneys were enlarged, softened, and partially disintegrated, presenting an olive-green color. The fat, and also all of the internal viscera, presented an icteric appearance. I recommended a change of pasture and water; the hog-pens to be removed and the boards used for some other purpose; the old hog pasture to be plowed up, and seeded with wheat or other grain for at least two years before it should again be used for a hog pasture. I also advised a destruction of all the hogs that indicated plain symptoms of the disease, and afterwards an entire change in the breed or family of hogs. I inclosed in a tin can a portion of the lung, liver, intestine, and stomach of the pig examined, and sent it to your Department by express, for microscopic examination.

After my return to Wellington I saw Mr. Hamilton again, when he told me that he and his partner, Mr. Flint, lost 16 or 17 head of their cattle out of 900 head. Their pasture is fenced in, and is located in the corner of Kingman, Pratt, and Barbour Counties, and comprises 30,000 acres. This pasture held Texan cattle last year. The cattle this year in the field were not exposed to other cattle. The disease appeared in the month of September; 10 or 12 that were sick recovered. Changing pasture and water seemed to offer a check to the disease.

I returned to Harper in the night. On the 23d it rained hard all day, and I remained in town. I saw I. J. Campbell, esq., who gave me the following history: He owns a fenced pasture on the western edge of town. On the 10th day of April Mr. Boyd placed 14 head of lame cattle in this field, which already contained 150 head of natives owned by Mr. Campbell; the Boyd cattle remained for a week or more to recuperate, and were then driven by the way of Pratt's to the Boyd range. Some time during the month of July 2 steers that were brought from the northern part of the county were turned into this field; soon thereafter both sickened and one of them died. The steers in coming to Harper crossed a number of cattle trails. None of Mr. Campbell's cattle died. Mr. R. A. Jones, from Labette County, told me that while on a visit to his brother at Santiago, Cal., last year, his brother told him that cattle that were brought from Old Mexico and driven north into colder and freezing climates in California impart disease to native cattle.

October 24 we drove southwest 16 miles to see Mr. Richard Botkin. He held cattle belonging to Shafer, Coyer, Arnold, Collins, McMullen, and Martin, the total number being 562 head. All of them were native cattle except the McMullen cattle, which were brought from Webster County, Missouri, reaching Botkin's range in the month of May, and remained until the 1st of September. Dr. Wisner took away 120 head of them, and the remainder were sold to and taken away by Mr. Potter. The Boyd cattle passed over his range in April, and all of these cattle have been grazing over their trail. All the deaths in this herd which occurred while on the Botkin range took place between the 2d of July and the middle of August. Loss, 184. Mr. Campbell, at Otega, lost one work ox out of a yoke of oxen in the month of July.

Munger Brothers, of Harper, started from Harper in the month of May 10 thoroughbred and high-grade bulls; by mistake the driver turned them in with the Boyd cattle, where they remained over night. Seven of these died, and 3 others in the herd of 500, on the Hink Moore range. They began to die sixteen days after exposure to the Boyd cattle.

October 25, we drove 7 miles west from Harper, stopping first at Mr. H. F. Burr's place. Mr. Burr stated that he shipped into Harper, then drove to his place, four lots of cattle; the first one in the month of March, one in April, one in May, and the last one in June; total number, 450 head. These cattle were brought from Colony, Anderson County, and from Coffee and Allen Counties. Mr. Burr wintered 18 head of cattle. He has been on the same range for five successive years, and has never before lost any cattle. He lost 175 head out of the 468 head this season, and Mr. Moier, his son-in-law, lost on the same range 4 out of 7 head. Mr. Burr thinks that all of his cattle except 25 head had been sick. He used tincture belladonna as a curative agent. Several of his cattle died in the month of July, but the greater number of deaths occurred in the month of September. Mr. Boyd's cattle crossed his range in April. We went out to his herd and I tested the temperature of several, which registered as follows:

	°F.
Two-year-old heifer, has been sick	102.7
One-year-old heifer, has been sick	104.2
One-year-old heifer, has been sick	104.8
One-year-old heifer, has been sick	104.2
Four-year-old bull, has not been sick	102.9
One-year-old heifer, has not been sick	103.4
One-year-old heifer, has not been sick	103.1
Two-year-old heifer, has not been sick	102.9

We then drove 1 mile southwest to A. Hilliard's farm. He lost 1 cow on the 20th of July, and another on the 10th of September. Mr. Boyd's cattle crossed his range half a mile south of the house. On the 18th of June Mr. Anderson's cattle went across the east end of his range, leaving probably 80 rods between the two trails. Hilliard has owned a work ox for the past two years. This ox was picketed on the Boyd trail a number of times during the summer, but remained well. While I was there I noticed a bull corraled near the barn; he appeared thin in flesh, and I inquired if he was sick. Mr. Hilliard replied, not that he was aware of. I tested the temperature of the animal; it registered 102° F.

An eight-year-old cow was in a separate pen; I tried her temperature; it was 101.8° F. On the 30th, as I was going toward Harper in the evening, I met Mr. Hilliard. He stopped me and stated that the eight-year-old cow which I saw in the pen at the time of my visit to his place was sick. "She appeared stupid last night, and this morning yielded no milk." That this cow had positively not been exposed to the Boyd trail, or any other infected place; that she had been kept closely yarded, and had been well fed on dry feed for the past three months. I promised him to see her in the morning. The following morning I drove out to his place, and found her manifesting the following train of symptons: Found her lying on the sternum, head well poised; horns warm; nose dry; a dull and drowsy appearance of the eyes; sensible to the flies; pulse depressed; respiration 22; temperature 104° F.; passage of manure slightly covered with mucus, and of a firmer consistency than it should be. I learned that this cow had been turned out to graze with the other cattle on the 29th, and as she had been kept up on dry feed for the past three months I concluded that she had overcharged herself with food to which she was not accustomed, and that this was the cause of her disability. On the 30th she would neither eat nor drink, but was disposed to lie down all the time. When I saw her on the morning of the 31st, she drank half a pail of water, and ate a few ears of corn; therefore I concluded that she must be improving; that she was better the day I saw her than on the day previous. On the 3d of November I again saw Hilliard, in Harper; he reported that the cow had recovered her health.

From Hilliard's we went to Matt. Miller's, 5 miles northwest of Harper. He stated that in the month of July Hugh McClung brought from north of Little Rock, Ark., 65 head of cattle; that he bought 35 head of these cattle from McClung, and placed them with 40 head of his domestic cattle on the same range; he lost 9 head, while McClung, who held the remaining 30 head on a range of Mr. Matthews, lost none. His cattle died during the first and second week of September, after a sickness of from two to five days. Miller sold 30 head of his cattle on the 10th of September.

Messrs. Got and Weaver, of Springfield, Mo., brought into Harper on the 24th of June, 200 head of cattle from Missouri. They drove them out 3 miles northwest, and had them herded on the Matthews range. About the 20th of July deaths among this herd began to occur, and they lost 24 head of their cattle in rapid succession.

October 26, we left Harper for Medicine Lodge, Barbour County, a distance of 35 miles. We arrived toward evening. I saw several cattle-men in town; made the usual inquiries, and was told that many cattle died during this season all over the county; but, when I endeavored to obtain a more definite account as to who the suffering parties were, and the extent of their losses, I could get but little reliable information. I concluded to remain in town over Saturday the 27th, and endeavor to get a more definite history of the outbreak in this part of the county. In the evening I was introduced to Mr. Ebersole, who lives 7 miles west of Medicine Lodge. He told me that on a range next to him 10 had died out of 700 head during the month of September, but that he, himself, had not lost any out of the 50 head which he owned. I also met Mr. Springer, who lives 5 miles southeast of Medicine Lodge. He lost 4 out of 420 head this summer, and 125 out of a herd of 500 last winter. Mr. Springer also stated that his neighbor, Mr. Bullington, lost 12 out of 100 head this season.

On October 27 I went to the office of Mr. T. L. O'Bryan, a live-stock broker. He told me that some emigrants passed through the town of Medicine Lodge, going north, on the 23d or the 26th of May. They were supposed to come by way of Anthony, Harper County, and were going to Colorado. They had with them 4 or 5 ox-teams, with from one to three yoke in a team, and 50 head of loose cattle. They told Mr. Riggs, the sheriff of Barbour County, that they came from Texas, but told Mr. O'Bryan that they were from the Chickasaw Nation, near Red River. Their cattle were in good condition and had the appearance of genuine Texans.

In Mr. O'Bryan's office I entered into conversation with Hon. T. J. Shepler, who told me that in the year 1876, when the through trail for Southern cattle to Dodge City passed along the eastern banks of the Elm River, near Medicine Lodge, 2 head of domestic cows which had been kept in close confinement all summer, were allowed to graze over the trail late in the fall after a fall of 3 inches of snow, and after so late an exposure both cows died. He also told me that a prominent stockman from Montana assured him last spring that cattle coming from the State of Kansas would transmit to their cattle the Texas fever in the most malignant and fatal form. Mr. Shepler thinks the only way to obviate the annual losses among cattle in Kansas by the Southern fever, is to establish a border line of infection and compel the Southern cattle to be slaughtered within the limits of such boundary line.

I then went to see Mr. Frank H. Shelley, secretary of the Salt Forks and Eagle Chief pool. He said:

> A number of through cattle, purchased at Caldwell, were located on the Eagle Chief Creek adjoining us on the southeast; some of the Salt Forks cattle drifted on the "T

5" range, belonging to the Texas Land and Kansas City Company; several of our cattle died, but not many; two of them were high-grade bulls. I don't know how many of our cattle were exposed. The pool holds 20,000 head of cattle.

I next met Mr. J. A. McCarty, in the office of the Barbour County *Index*. He is the captain of the Sand Creek and Hackberry pool. He stated that in the month of August, Mr. Lockhart, a member of the pool, brought from Kingman County several hundred head of cattle and placed them in the pool herd. Three weeks thereafter the pool cattle began to die; 15 out of 3,500 head exposed died It is supposed that several head of Arkansas cattle were among the Lockhart lot. Ten per cent. of their cattle died last winter. I met Mr. William Kelley on the street; he lived 8 miles south of Medicine Lodge. He lost 10 out of 400 head this summer. He does not know in what manner they were exposed. Last winter he lost 50 out of 500 head; they generally became lame in one fore leg; persisted in lying down; would continue in this way from one week to a month before they died. Many of them were valuable cows. They were well sheltered and well fed after they became sick, but the majority of them ultimately died. Several died after the grazing was good in the spring. In 12 or 15 cases that recovered one or both horns came off. Mr. Vaughn, 2½ miles south of Medicine Lodge, brought 60 head of cattle from Kingman County about the 1st of July. During the month of September he lost 3. Last winter he lost in Kingman County 42 head out of his herd of 300. The best cattle in the herd appeared to be the most susceptible. His cattle were fed on corn in the latter part of the winter. James Wilson, 8 miles south of Medicine Lodge, stated that he gathered one bull at the county round-up which died.

Two other animals died subsequently, and he does not know where these were exposed, except to the bull. Lost last winter 6 per cent. Mr. Hamlin, neighbor to Wilson, lost out of 3 head gathered on the Boyd range, one bull. Mr. B. D. Keyes, on Elm River, 18 miles northwest of Medicine Lodge, reported a loss of 40 head out of 500 last winter. He fed millet, hay, and corn. They were sick from two days to two weeks. Some got lame, and would then lie down nearly all the time; most of them ate well until they died. One cow ate two quarts of soaked corn, then dropped over dead. He further stated that this trouble was almost universal in that part of the county. After skinning the dead cattle he found infiltrations of bloody water under the shoulder blade and foreleg. Mr. B. T. Shields, a neighbor to Keyes, lost this summer 16 out of 225 head.

On October 28th we left Medicine Lodge and drove west toward Lake City; the first place at which we stopped was Henry Morehead's, 3 miles west of Medicine Lodge. He told me that he had 144 head of cattle in a herd, under the care of P. B. Cole, on Antelope Flat, northwest and north of the Illinois colony. Also, that his neighbor, Mr. Updegraf, had 60 or 70 head in the same herd; neither of them had suffered any losses. The next place on our way to Lake City at which we stopped was Mr. Sanderson's, who lives on the north side of the Medicine River, 8 miles west of Medicine Lodge. He lost 3 head of cattle out of 40 in this month. Cause of death, or manner of infection, not known. He told me that Mr. Robert Ingram, on Cedar Creek, 7 miles west of Medicine Lodge, lost 6 out of 50 head during this month. Manner of exposure not known. We then proceeded to Lake City, which is 18 miles northwest of Medicine Lodge, and from there we drove 2 miles north, to Mr. W. F. Gordon's ranch. Mr. Gordon holds 400 head of cattle on a 7,000-acre fenced range. This range is supplied with water from several clear-water springs, all of which head within the inclosed range. Mr. Gordon lost 35 head of cattle. The first one died on or about the 25th of September, and the last one on the 25th instant. Ten or 12 that were sick recovered. Mr. Gordon knows of no way in which his cattle were exposed, only that once or twice the gate at the north end of the field was left open by persons passing through, and a few of his cattle got out, but were always returned within a few hours. One hundred and eighty-nine head of these cattle were bought from Reuben Lake, of Lake City, on the 1st of July. They were Arkansas cattle, wintered by Mr. Lake in a fenced field adjoining that of Mr. Gordon. The balance of Gordon's cattle, 212 head, were double wintered in his field. As I was anxious to make a *post-mortem* examination in order to discover the exact nature of the disease, I went out late in the evening to see the condition of the heifer which died on the 25th, three days ago. She was badly bloated. I opened her in the usual manner, and found that the internal viscera were undergoing decomposition, and were scarcely fit to handle. I removed the spleen, which weighed 4 pounds. The liver was enlarged. A bloody-colored fluid was contained in the pericardial sac; also bloody-colored urine in the bladder. The fourth stomach presented the characteristic erosions and gastric redness of southern cattle fever. Mr. Gordon stated that in 1858 he lived in the State of Missouri, on the through cattle trail. The trail led around a corner of his pasture field, the corner not being quite square; late in the fall he built the fence out so as to form a square corner; by so doing he inclosed a part of the trail. The cattle which were in the field soon began to die after the moving of the fence, and continued to die until some time in the month of January; 50 out of 100 head died.

We remained over night with Mr. Gordon, and next morning returned to Medicine Lodge. As we passed Mr. Morehead's place his wife came out to the road and told me that her husband had found the day before a sick heifer among his cattle on the Cole range; that he had started her toward home, but she dropped down near Elm River, and was unable to rise, and that he desired me to see the animal. I was directed where to find it, and I complied with their wishes; I found the heifer dead. I saw the animal at 2 o'clock on the 29th, and Mr. Morehead had found her sick at about the same time the day previous. She appeared to me as if death had taken place in the night or early morning; was badly bloated; a few small balls of feces, covered with dried blood, and mucus, were lying behind her. The mucous membrane of the rectum appeared inflamed, swollen, and protruded several inches from the anus. I opened her on the right side, following the usual custom. Decomposition had taken place to a much greater extent than any that I had ever examined after so short a period after death.

The blood-vessels contained more blood, and of a better quality than is usually found after death from southern cattle fever. The animal was excedingly fat, and the fat was of a very high yellow color, very nearly a chrome yellow. All of the internal viscera were, more or less, distended by gasses. The pericardium contained 10 or 12 ounces of bloody-colored serum. In the arterial side of the heart I found a very extensive and strongly organized fibrinous clot, extending through the valves, and into the aorta for at least 8 inches. The spleen weighed 3½ pounds, and blood extravasations were found under the peritoneal covering. The liver was enlarged, and filled with blood, and enormously distended by gasses. Erosions and subacute congestion of the lining membrane of the fourth stomach were present. The urine bladder contained a few ounces of bloody-colored urine. The kidneys were almost completely disorganized by the ravages of active decomposition.

At Medicine Lodge I saw Mr. Standiford, of the cattle firm of Standiford, Youmans & Co. Their range is located 6 miles southeast of Medicine Lodge, between the Medicine River and Cedar Hills. He made the following statement:

> About 150 head of cattle, coming from near Wichita, Sedgwick County, were driven along south on the divide between Antelope Flat and Elm Creek, and were brought to and camped on the center of our range one night. As near as I remember the date it was about the middle of June. I went out to see the cattle; they looked very suspicious. Mr. Shanstrom, who was with the cattle, told me that the cattle came from Arkansas; that he bought them in February, and shipped them into Wichita about the 1st of April; that they were kept on rough feed, and afterward herded on the Ninnescah River until they were started South. Several well-bred bulls, which were bought at Wichita, were among the herd, but a large proportion of the herd looked bad. From my place they went southwest 100 miles to the Cimarron River, and were placed with some other cattle which were in a small pool formed by Blackstone, Tucker, Mills, Conner, and Shanstrom.
>
> About one week after Shanstrom's cattle left my place Mr. Tucker (of the above-mentioned pool) came to my place and told me that the Arkansas cattle which Shanstrom took down were dying off rapidly; also, that some of the other cattle in the pool were dying. On the range where these cattle camped one night we have 700 head; they have been close ranged in the spring and summer, but several of them drifted away; afterward 3 were gathered on the Boyd range at the time of county round-up. Two weeks after the Shanstrom cattle were on our range one of ours died; soon another one; the second one was gathered on the Boyd range. Texan cows which had been double wintered, and had also been exposed to the Boyd cattle, remained well. One bull died that I kept up and stall-fed all winter, which I know had no chance for exposure to the Boyd cattle, nor did he stray away from our herd. He died in the first week of October. Another bull which had been kept exactly like the first one died on the 25th. One cow that I kept in town until the last of June and then took her to the herd, where she was kept within sight every day, also died. Altogether we lost ten of the very choicest cattle in the herd. Last winter we lost 10 per cent. of our domestic cattle, and 6 per cent of the Texan cattle.

I met Mr. A. L. Duncan, of Medicine Lodge, who told me that three different herds of cattle passed through their place in the latter part of May and early June. One of the outfits told him they were from Red River, Chickasaw Nation, and said they were going into Colorado. The second outfit were going to Montana, and the third into Utah. The three herds numbered about 500 head of cattle, and looked like Indian or Northern Texans. From Medicine Lodge they passed in a westerly direction toward Fort Dodge.

While at Medicine Lodge I met Mr. P. B. Cole. He lives 2 miles west from Dr. Wisner and north of T. B. Stockstill. He owned 250 head of cattle, and held 150 belonging to other parties. These cattle were nearly all gathered in the county round-up on Antelope Flat, between the 1st and 15th of July, where they had been with several hundred other cattle. In this round-up 25 or 30 stray cattle were found for which no owners appeared. The Anderson herd of cattle grazed on the Cole range for several days in the latter part of June. Mr. Cole lost out of his herd of 400 25 head—2 in July and 23 in September. William Dark, north of Cole's, lost 16 out of 350 head of cattle during the month of September.

The first place at which we stopped, after leaving Medicine Lodge, on October 30, was the Hulitt, or open A range, on Camp Creek, Harper County. Hulitt Brothers brought 250 head of good high-grade cattle on the range from Iowa a year ago, which have remained on the same range up to this time. Mr. Miller, of Washington County, Iowa, unloaded at Harper, on the 9th of April, 220 head of Iowa cattle, kept them on the range north of the stock-yards and in the yards at night for three successive days, then drove them out on the range occupied by Hulitt Brothers, and placed them under their care. Mr. Ament, of Anthony, Harper County, shipped from Coffeyville, Mo., and unloaded at Harper 300 head of cattle; they arrived at Harper on the 28th of June. He then drove them southeast into the eastern part of the county on a school section, and herded them there until the 10th of July. They were then taken west and placed on the Hulitt Brothers range, arriving on the 12th. One of these cattle died on the same day that it arrived upon the range, and others of the Ament cattle died daily thereafter, until the number of deaths reached 47. None of the Miller nor of the Hulitt Brothers cattle died.

The last death among the Ament cattle occurred on the 1st of October. This was a black bull, bought in Kansas for $540. Forty head which had been sick recovered. The sick were treated by giving one quart of raw linseed oil at a dose, and by using the same by injection into the rectum. Some recovered after passing bloody urine. The Boyd herd of cattle ranged over this ground in the early part of the summer, and 3 head of them were with the Hulitt Brothers cattle later in the season, 1 remaining six weeks.

The Miller cattle, which were unloaded at Harper on the 9th of April, were with 20 head of the crippled Boyd cattle in the yards and on the range north of the yards, and ate corn, cane, and millet-hay from the same piles. The description of the *post mortem* appearances in the Ament cattle, as given me by Mr. Hulitt, convinces me that they died with the southern cattle fever.

We next stopped at Attica post-office, Harper County. There I saw Mr. G. W. Markham, who stated:

> One of my two-year-old steers got among the Boyd herd as they were passing along north of my range on the 10th of April. I followed him and brought him back home the same evening. Two days thereafter I sold him to H. D. Drumm, of Kiowa, who has since told me that the steer died.

Mr. Drumm had 2 head of the Boyd cattle among his herd from the latter part of April until the 1st of July, but did not lose any of his cattle except the one bought from Mr. Markham. At Attica I was told that a yoke of oxen had been staked on the Boyd trail, south of Gardner's; they were used for breaking sod, and remained there all summer. No sickness followed the exposure. In the evening I met Mr. J. C. Fox, of Seymour, Iowa, at the Glenn House, Harper. He told me that he brought four car loads of cattle into Harper on the 12th of April. He held his cattle in the yards at night and on the range north four or five days, 1 of the Boyd cattle being with them all this time. These cattle were taken northwest 18 miles, on a range, and remained there until the 20th of August. No loss or sickness appeared in this herd. Mr. Hardwick, of the Glenn House, told me that he had a sick cow, also that he lost one several days ago. I promised to go out in the morning to see her.

On the 31st I drove out 2 miles west to Mr. Forrey's inclosed pasture field to see the Hardwick cow. I found the cow in the following condition at 9.30 a. m.: Temperature 106°; lying on the left side with head thrown somewhat to the right; was apparently suffering great pain, as was manifested by her deep and agonizing groans; eyes prominent; left horn much colder than the right; nose dry; skin a deep yellow color; pulse 86; respiration 20; painful and prolonged expirations; could feel through the abdominal walls posterior to the ribs decided enlargement of the liver; auscultation and percussion revealed no lung trouble; thick viscid saliva stringing from the mouth—not profuse. At 10.15 the temperature was 97.5°. At 10.30 I introduced the instrument into the bladder, where it registered 97.4°. I cut a deep gash into the tail, 1 inch in length, about 4 inches below the root, in order to observe the flow and the color of the blood, but only a few drops oozed from the incision; it was very thin and watery. I cut another gash into the fleshy part of the thigh, but only a slight trickling of blood followed. In making these incisions the animal evinced no pain. At 11 o'clock she made an effort to get up, but failed. A sweat now broke out on the nose; pulse very tense, yet weak. As I stood by her side I could hear distinctly each heart beat. It appeared as if nature was concentrating all the strength that was within the animal to maintain the heart's action. She is now resting on the sternum, with head extended, the lower jaw resting on the ground, and groans at each expiration of breath, to which it is painful to listen. Tremors of the vasti muscles, and also of the muscles of the neck, now appeared. At 11.15 temperature 98°. I then left her; returned again at 2 p. m., and found her dead. The surface of the body was yet warm, and out of curiosity I inserted the thermometer into the rectum, when it registered 103.5° F. An hour later I returned prepared to make a *post mortem* examination. There were present at the examination Messrs. Ewell, Cochran, and Ross, of Harper.

The animal was lying on the left side, and a quart or more of a greenish watery fluid had escaped from the mouth and nose. After exposing the internal organs to view I found the lungs slightly emphysematous and a frothy sputa in the capillary tubes; pericardium contained about 6 or 8 ounces of dark, bloody-colored fluid; external surface of heart extensively ecchymosed; in fact, looked limp, bruised, and worn out by sheer exhaustion; the internal surface of the heart was almost black, caused by capillary congestion and extravasation of blood into the endocardium; no blood clots in the heart. The heart weighed 5 pounds. The spleen weighed 4 pounds, and presented a disintegration of the glandular structure; the external surface presented numerous ecchymosed spots, and a purple and gray-mottled appearance. The liver weighed 15 pounds, and possessed a spongy feel to the touch; was darker in color than normal, and manifested fatty degeneration. The gall bladder contained 30 ounces of thick, granular appearing bile, of a greenish-brown color. The third stomach presented nothing abnormal, but the fourth presented the characteristic redness and erosions, exposing the vascular membrane, indicative of southern cattle fever. Surrounding the kidneys was discovered a yellowish gelatinous infiltration. The kidneys were darker than normal, and contained bloody-colored urine in the ducts and tubuli. The uterus contained a six-weeks' old fetus; this organ presented no marked lesions. The urine bladder was distended with 2 gallons of a dark, almost brown, colored urine; specific gravity 1.012. The blood in the blood vessels was not so thin as is usual in such cases, but was deficient in quantity. Mr. Hardwick bought these two cows, together with 48 other cattle, on or about the 10th instant, from Mr. Bailey, who lives 2 or 3 miles northwest of town. The 48 head were taken down into the Indian Territory on the 16th—the same day the two cows were placed into the Forrey field. All of these cattle crossed several cattle trails before they reached the Forrey pasture. This day I saw Mr. L. C. Bidwell, of Anthony. He owns 2,000 head of cattle, which are pastured in an inclosed field containing 12,000 acres, located in the Indian Territory, along the south line of Harper County.

He stated that cattle had been dying all around his pasture, but that he did not lose a single one. Mr. J. W. Walcott, of Harper, kept 11 cows from which he supplied milk to the citizens of Harper. He herded them north of the railroad track. The first loss occurred on the 4th day of July, after six days' sickness; 10 head died before the 1st of August. The remaining one was sick three or four days, then began to improve, and in a week was again apparently well. Mr. R. J. Jones picketed his cow north of the railroad track for the space of a week in the early part of July; no sickness followed. He has owned the cow for four years. John Elrod, of Harper, owned 8 head of work oxen, all of them wintered cattle. One of them was a Texan, eight or ten years old, and had been owned in the county several years. These cattle were pastured north of the railroad at Harper for a month before any disease appeared among any of the cattle in or around Harper. In the month of October 4 out of the 8 oxen died, the old Texan being the last one to succumb to the disease.

On November 1 we drove to Anthony, 9 miles south of Harper, principally for the purpose of seeing Mr. Ament, who suffered such a heavy loss on the Hulitt range; but we failed to find him at home. Here I made inquiries relating to disease among cattle, and was told no cattle in that immediate locality died this year. Mr. Northup, a prominent cattle man of Anthony, told me that nearly all the domestic cows along the trail of the Anderson cattle died. The Anderson cattle passed 6 miles west of Anthony on their way south to the Territory line. He also told me that Mr. Singer, 10 miles east of Anthony, lost a large percentage of his cattle last winter; they are said to have been in good condition and were well fed. They had been brought from Iowa and Northern Missouri in the fall. After returning to Harper I traced up the town cows that died in Harper as follows:

Owners.	Number of cows lost.	Value.
J. M. Bloom	2	$70
John Grimes	1	40
C. Arthur	2	75
R. Gorman	1	40
Mr. Schwenk	1	35
P. P. Thomas	1	36
E. Keefer	1	35
George Cook	1	35
John Hyatt	2	80
M. Cochran	2	90
B. Orange	1	40
J. Burd	1	35
Mr. Cyphers	1	38
S. Keever	1	46
Total	18	695

All of these cows grazed north of the railroad track. The town cows which were not allowed to graze north of the track escaped the disease.

HISTORY OF THE ANDERSON CATTLE.

Six car-loads of cattle shipped from Springfield, Mo., in the name of the Bank of Springfield, and consigned to Mr. Anderson, were unloaded at Harper on the 6th day of June. These cattle remained at the stock-yards about ten days, ranging northeast of the yards during the day. They were then taken out west about 4 miles, southwest until they reached the Medicine Lodge road, followed this road into Barbour County, through the Illinois colony, and rested upon the range of P. B. Cole a week or more. They then were driven back again by way of Inyo post-office, thence by Joppa post-office, passing over W. E. Kline's range 5 miles west of Attica post-office, then in a southeasterly direction toward Anthony, but leaving Anthony 6 miles to the east, then south to the line of the Indian Territory, then east along the line to Gilmore's range, 6 miles west of Caldwell, in the Indian Territory. Seventy-eight head of these cattle were sold before they left Harper to a Mr. Smalley, in the southwest corner of Kingman County, and were driven by him in a direct course northwest from Harper to Kingman County, Mr. Anderson stated that these were Arkansas cattle. Mr. Cochran stated that he bought conditionally 100 head of cattle in White County, Arkansas, last winter, but that Mr. Anderson afterward saw the same lot of cattle, and by offering more for them closed a bargain, and drove them to Springfield, Mo.

On November 2, we drove out to see Mr. Potter, but he not being at home his son gave me the following information: They held cattle 12 miles northwest of Harper; they bought 131 head of cattle out of the McMullen herd on the Botkin range; afterward added 106 head of domestic cattle brought from the line of Kingman County. Two native milch cows were also turned in with this herd. Out of the first herd (131) 32 died; out of the 106 head 26 died. All of these, excepting 4 head, died previous to the 10th of August—the 4 died in September, and 10 that were taken sick in September recovered. The first lot of cattle were bought on the 1st of July, and were taken on the range on the 8th; the second lot were bought a few days later. The McMullen cattle began to die on the 8th of July. Mr. Potter knows of no other cattle dying near their range.

Clotfelter and Thomas have a fenced pasture, 6 miles square, in Kingman and Harper Counties. This field contained cattle belonging to Clotfelter and Thomas, 300 head; Aaron Canalt, 160 head; Mr. Blake, 300 head; Harroldson and Sheldon, 700 head. All of these cattle, except 640 head of Harroldson and Sheldon's, were wintered in this field, and during the winter 150 head died, the loss being attributed to insufficient food and water, and want of shelter. Harroldson and Sheldon placed into this field, in the month of June, 700 head of cattle, said to have been brought from Missouri; Munger Brothers put in 400 head after the disease broke out, and allowed them to remain until the 1st of October. In the month of July disease appeared among cattle in this field, and Harroldson and Sheldon lost 30 head. They removed their cattle early in September. The loss among Blake's cattle I could not ascertain. Clotfelter and Thomas lost 3 head; Munger Brothers, 16. No other cattle adjoining this field died, except a few in Flint & Hamilton's field, located in the corner of Kingman, Pratt, and Barbour Counties.

William Nance, 15 miles northwest of Harper, bought 2 steers out of the Boyd herd in the spring, took them home, and picketed them near his house. After the steers were removed a cow was picketed on the same ground; in about two weeks she became sick and died. Soon afterward Mr. Nance bought two cows, picketed them on the same place; both of them became sick, and one died. The steers remained well. This completes my investigation of cattle disease in the counties of Harper and Barbour.

List of herds of cattle suspected of conveying the disease.—No. 1, the Boyd cattle, arrived at Harper April 6; No. 2, the Anderson cattle, arrived at Harper June 6; No. 3, the Scoby cattle, arrived at Harper June 6; No. 4, the McClung cattle, arrived at Harper July; No. 5, Arkansas cattle, which crossed McGee's range in June; No. 6, three emigrant herds through Medicine Lodge June 1; No. 7, Shanstrom cattle, June 1; No. 8, Garrison cattle, arrived at Harper June 18; No. 9, county round-up, in Barbour, July 1 to 15; No. 10, Territory round-up, in Barbour County, June and July; No. 11, emigrants which passed by Pratt's in June; No. 12, the Wisner oxen, *including the Downing yoke*, arrived at Harper in February; No. 13, Martin cattle, from Mr. Potter, October 6.

It may be said that all of these herds rest under greater or less suspicion; but, owing to the limited time allotted to me for my investigations, I have not been able to establish the fact that any of them were capable of communicating disease to other cattle, directly or indirectly.

The following summary may be readily understood:

Owner.	Exposed to herds number—	Number of cattle exposed.	Number sick.	Number of deaths.	Number of recoveries.	Date of outbreak.	Value.	County.
Dr. Joseph Brockway	1, 11	75	28	23	5	July 10, Sept. 12.	$575	Harper.
E. Walden	1, 2	7	7	7	......	Sept	275	Do.
William A. Wood	1, 2	13	5	1	4	Oct. 12	30	Do.
Jesse Boyd	8, 9, 10, 12	680	16	16	......	July 10, Sept.	820	Barbour.
R. B. McGee	5, 9, 10	200	29	21	8	Sept. 15	630	Do.
W. W. Cook	1, 5, 7, 9, 11, 12	831	90	75	15	July 9, Aug. 29.	2,000	Do.
A. B. Ranalls	1, 5, 7, 9, 11, 12	150	30	30	......	Sept	900	Do.
John Elmore	1, 5, 9, 10, 11	250	156	156	......	July 1, Sept. 1.	4,680	Do.
R. J. Evans	......	116	10	6	4	Oct. 3	800	Do.
Mr. Parsons	5, 6, 9, 10	80	5	5	......	Aug., Oct. 20.	200	......
Hink. Moore	5, 6, 9, 10	700	5	5	......	......	150	Do.
Thomas Brakey	5, 6, 9, 10	200	15	15	......	Sept. 15	600	Do.
C. Blackstone	......	110	4	2	......	Oct. 12	80	Do.
W. E. Campbell	Fenced	3,500	7	7	......	......	210	Do.
Tryor & Miller	do	500	10	10	......	Sept, 10	300	Indian Territory.
Mr. Latham	do.	500	16	16	......	......	480	Barbour.
H. Hale	1, 5 6, 9, 10	300	53	33	20	Sept. 15	990	Do.
John Peters	1, 9, 10, 11	255	136	96	40	July, Sept. 1.	2,280	Do.
David Clough	1, 8, 9, 12	300	77	67	10	July 4, Sept. 1 to 10.	2,450	Do.
E. C. Davis	1, 2, 8, 9, 12	60	9	6	3	July 1	236	Do.
William Garrison	1, 2, 8, 9, 12	600	185	85	100	July, Sept. 10.	2,250	Do.
Mr. White	1, 2, 8, 9, 12	10	3	3	......	Sept	120	Do.
Mr. Pelton	1, 2, 8, 9, 12	20	2	2	......	July, Sept	70	Do.
T. B. Stockstill	2, 8, 12	150	5	2	3	Oct. 6	90	Do.
M. B. Moore	1, 2, 8, 9, 12	123	48	40	8	July 11, Sept. 15.	2,000	Do.
M. McGuire	1, 2, 8, 9, 12	60	6	6	......	......	240	Do.
B. F. Kemp	1, 5, 6, 11, 12	108	30	15	15	Aug	480	Do.
[illegible]. E. Mattox	1, 9, 10	125	7	7	......	July	245	Do.
James Roberts	1, 2, 8, 13	17	1	1	......	Oct. 6.	20	Do.
Mr. Reeder	1, 9, 10	27	3	3	......	July	100	Harper.
W. H. Wisner	1, 2, 11, 12, 13	120	30	30	......	Sept.	600	Barbour.
	7, 8, 12	300	3	3	......	Oct.	500	Do.
J. H. Warren	1, 2, 8, 12	40	4	4	......	Sept.	160	Do.
Mr. Gardner	1, 2, 11	3	1	1	......	......	40	Harper.
L. M. Pratt	1, 11	15	7	7	......	July 4	340	Do.
F. P. Melvin	1, 2, 11, 13	4	2	2	......	Oct. 8	84	Harper.
C. P. Bradford	......	6	6	5	1	Oct.	205	Do.
Louis Hildebrant	13	10	10	3	7	Aug. 20	120	Do.
S. H. Coyer	1, 2, 11	18	14	14	......	July, Sept	560	Do.
S. M. Shafer	1, 2, 11	18	10	10	......	July, Sept	800	Do.
E. Vian	1, 11, 13	100	36	36	......	Sept	1,260	Do.
A. T. Woodward	3	56	44	36	8	Sept. 1	1,270	Do.
John Challis	3	100	18	12	6	Sept. 1	400	Do.
Mr. Seoby	1, 2, 4, 8, 12, 13	200	10	10	......	......	250	Do.
W. A. Creighton	1, 11	27	1	1	......	July	200	Do.
T. Goss	......	70	60	60	......	......	1,800	Linn.
Mr. Donahue	......	14	2	2	......	Sept	70	Sumner.
Flint & Hamilton	......	900	29	17	12	Sept	950	Kingman, Pratt and Barbour.
R. Botkin's herd	1, 2, 11	562	184	184	......	July 2	5,520	Harper.
Mr. Campbell	2, 5, 11	2	1	1	......	July	50	Do.
Munger Brothers	1, 2, 5	510	10	10	......	May or June	750	Barbour.
H. F. Burr	1, 2, 13, 12	475	450	179	271	Sept.	3,495	Harper.

Owner.	Exposed to herds number—	Number of cattle exposed.	Number sick.	Number of deaths.	Number of recoveries.	Date of outbreak.	Value.	County.
A. Hilliard	1, 2	5	2	2		July, Sept	$80	Harper.
Matt Miller	4	75	9	9		Sept	200	Do.
Got & Weaver	1, 2, 3, 8, 12	200	24	24		July 20	600	Do.
Ebersole's report		700	10	10		Sept	300	Barbour.
Mr. Springer		420	4	4			125	Do.
Mr. Vaughan	6, 7	60	3	3		Sept.	75	Do.
Mr. Bullington		100	12	12			400	Do.
Salt Forkes and Eagle-chief pool.	(*)	20, 000		several				Do.
Land Creek and Hackberry pool.		3, 500		15			500	Do.
William Kelley	5, 6, 7	400	10	10		Sept.	300	Do.
James Wilson	6, 7, 9	550	3	3			150	Do.
Mr. Hamlin	1, 6, 9, 12	3	1	1			160	Do.
B. F. Shields		225	16	16			500	Do.
Mr. Sanderson		40	3	3		Oct	120	Do.
Robert Ingram		50	6	6		Oct	210	Do.
W. F. Gordon		400	47	35	12	Sept. 25	1. 555	Do.
H. Morehead	2, 3, 12, 9	144	1	1		Oct. 29	30	Do.
Standiford & Co	1, 5, 6, 7, 9	700	10	10		July, Oct.	520	Do.
P. B. Cole	1, 2, 7, 8, 9. 12	400	25	25		July, Sept	1, 000	Do.
William Dark	1, 2, 7, 8, 9, 12	350	16	16		Sept	480	Do.
Mr. Ament	1, 2, 3, 8	300	87	47	40	July 12	2, 081	Harper.
"Markham steer"		1	1	1			30	Do.
Mr. Hardwick	(†)	48	2	2		Oct. 26	80	Do.
J. W. Walcott	(‡)	11	11	10	1	July 4	500	Do.
John Elrod		8	4	4		Oct	200	Do.
Town cows, Harper		18	18	18		July 4	695	Do.
Potter & Son	1, 2, 3, 8, 12	239	68	58	10	July 8	1, 160	Do.
Clotfelter & Thomas		1, 560	49	49		July	1, 470	Kingman and Harper.
William Nance		3	2	2	1	July	80	Harper.
Total		19, 229	2, 272	1, 768	604		$53, 756	

* Supposed Arkansas cattle.

† All of the trails at the west edge of Harper.

‡ All north of the railroad at Harper.

It will be safe to say that 2,000 head of cattle died this year, in Harper and Barbour Counties, with the southern cattle fever, and that the direct and indirect loss will not fall short of $75,400.

GLANDERS AMONG HORSES.

Before leaving Harper, I went northeast 11 miles to the residence of Mr. Alexander Cheesman, to investigate the nature of a disease among horses, supposed to be glanders. After arriving at the place, Mr. Cheesman led the affected horses out of the stable. The first one I examined was a ten-year-old horse belonging to Joseph Cheesman. This horse had a discharge from both nostrils, of a greenish-yellow color; considerable tumefaction across the external surface of the nasal bones; dullness upon percussion was manifest over the maxillary sinuses; numerous ulcers of various sizes were visible upon the nasal septum, chancre-like, and of a dirty yellowish color, with elevated serrated borders; milliary tubercles appeared in clusters on the schneiderian membrane, extending as far up in the nasal cavity as I could see. Both the submaxillary lymphatic glands were enlarged, hard, and nodular to the sense of touch, but not strongly adherent to adjacent structures. An abscess was in process of formation on the lower surface of the chest, one on the inside of the right hind leg, another one on the molar bone, and one on the left temporal bone—veritable farcy buds. On percussion, dullness was manifest over the inferior lobe of the left lung; respiration was accelerated. There was also a discharge of thick viscid matter from the left eye, and swelling and partial protrusion of the membrane nictitans. I was told that this horse had been coughing, more or less, for a year.

The next one which was led out for examination was a six-year-old bay mare, also belonging to Joseph Cheesman. She had a discharge from the right nostril, which was of a very gluey character, adhering around the margin of the nares, numerous small characteristic glanders ulcers on the septum nasi, and enlargement of the submaxillary lymphatic glands on the corresponding side. Both hind legs were edematous and presented swelling and tumors along the lymphatics, extending from the hock upwards to the inguinal region; the inguinal glands also were enlarged, hard, and sensitive to touch.

The third animal examined was a gray horse, fifteen years of age, belonging to Alexander Cheesman. He had a discharge from the left nostril; a hard swelling—the size of a walnut—of the left submaxillary lymphatics; a few small circumscribed tumors distributed over the surface of the body—farcy buds. This horse presented no visible nasal ulcerations. I examined two other horses, which have been in the same stable with the affected ones, but could discover no evidence of disease in either of them. Mr. A. Cheesman told me that a four-year-old mule died in the same stable in the month of March, and that she presented symptoms similar to the first horse that I examined. I pronounced the three horses to be affected with glanders and urged the owners to have them destroyed; but they did not promise to follow my advice, unless they could get some recompense from the county or State. On the 10th of November I addressed a letter to Hon. G. W. Glick, governor of the State of Kansas, stating to him how I found those horses affected, and requesting him (in the absence of a State board of health) to take the matter in hand if he had any authority to order the destruction of such diseased animals. In reply I received from him the following answer:

> I have no authority under the laws of this State to do anything in relation to the diseased horses of which you write, but I shall call the attention of the county attorney to the matter and see whether he can do anything by communicating with the parties to induce them to kill their diseased stock.

HOG CHOLERA, OR SWINE PLAGUE.

Having heard that hog cholera proved very fatal to hogs in the vicinity of Mulvane, Sumner County, Kansas, I left Harper on the morning of the 5th and arrived at Mulvane in the evening. There I saw Mr. E. F. Osborn. He informed me that Mr. Rucker, Mr. Kennedy, and Mr. Smith had suffered heavy losses this year by the death of their hogs. Next morning I saw Mr. A. A. Rucker, who resides three-quarters of a mile south of Mulvane. He told me that a year ago a number of hogs were shipped into Mulvane, coming from the State of Iowa. Several of them broke out of the yards and had the run of the town for several days. They got in with some hogs belonging to Mr. Hill, of Mulvane; soon afterward Mr. Hill's hogs began to sicken and to die. Mr. Rucker's hogs escaped from their pasturage and got in with Hill's hogs, rooted around, and probably ate of some of the dead; in seven or eight days Mr. Rucker's hogs became sick, and many of them died—he lost 25 per cent. Ten females recovered, and were kept until this spring, but failed to breed. Last spring Mr. Rucker bought 110 head of hogs from his neighbors, and placed them on the same grounds where the hogs had died the year previous. In the month of June they began to die; and this time he lost, including small pigs and shoats, 150 head. Ten of them were large, fat hogs. Estimate of loss, $800. I then saw Mr. E. A. Kennedy, who lives 3 miles south of Mulvane. He lost this year 150 out of 350 head of hogs, 50 of them being large heavy animals. Estimate of value, $1,000. Mr. Smith, a neighbor to Kennedy, also lost a large number of hogs this year. All the hogs in these three different herds have been indirectly exposed to the imported hogs, or to each other. From the description of the symptoms of the disease, as given me by Messrs. Rucker and Kennedy, I conclude that the disease has been true hog cholera; but none were sick or recently died, therefore no opportunity was afforded me to establish the nature of the disease positively.

OUTBREAK OF SOUTHERN CATTLE FEVER IN BUTLER COUNTY, KANSAS.

In your instructions of the 9th of October, you referred me to Senator P. B. Plumb, of Emporia, for information in relation to the locality of a disease among cattle in Butler County. I addressed a letter of inquiry to Senator Plumb, dated the 13th of October, and received the following reply:

WASHINGTON, D. C., *October* 20, 1883.

DEAR SIR: Yours of the 13th has just reached me. I do not know exactly whom to suggest that you call upon in Butler County for information about cattle disease, but if you call on Hon. A. L. Redden at El Dorado, or Hon. Neil Wilkie, at Douglas, they can put you on the track.

Respectfully,

P. B. PLUMB.

M. R. TRUMBOWER, V. S.,
Harper, Kans.

After the receipt of Senator Plumb's letter, I addressed the parties referred to and received answers from both, stating that I should go to El Dorado to find what I desired. I reached El Dorado on the 7th of November, and proceeded to Hon. Redden's office, but found him absent; his clerk took me to the bank of El Dorado and introduced me to Mr. V. Brown, who, in turn, accompanied me to the city mayor's office and introduced me to him—Dr. A. Bassett.

Dr. Bassett assisted me very materially in my investigations and manifested the greatest degree of interest in my work while I remained at El Dorado. At his office I was introduced to the Rev. S. F. C. Garrison, who resides 3 miles west of El Dorado. He made to me the following statement: That he placed under the care of Mr. Matthew Robeson 18 head of cattle on the 17th day of April—17 of them being grown cattle, the remaining one a calf; that all of these cattle, excepting 2 head, were to be kept in the dry herd; that on the 1st of June 1 of these cattle was taken away from the herd, and on the 1st of July a second one was removed, leaving 16 head (including the calf) to remain. On the 24th of September the 16 were also taken home; this took place owing to the report that cattle were dying in this herd under suspicious circumstances. When Mr. Garrison took his cattle home he turned them into a field with 12 head of other cattle. On the 30th day of September 1 cow died after a sickness of two or three days; 11 of them died in rapid succession, the last death occurring on the 10th of October. All of the 11 that died had been removed from the Robeson herd; none of the home cattle became affected. Mr. Garrison observed the symptoms manifested during the course of the disease to be—

> A peculiar odor arising from the skin of the affected animal; then a dry, hard, husky cough, especially when urged to move around; head carried extended; ears droop; pushing the head against straw-stack or fence; loss of appetite; no desire for water; rumination suspended; segregation; weak and staggering gait; saliva flowing from the mouth; whites of the eye assume a yellow tinge; perspire excessively toward evening, which is of a very disagreeable odor; shake the head from side to side as if in pain; trembling of the muscles sets in upon the slightest exertion; pulse beats rapid and hard; become unable to rise; partial coma and death ends the scene. One of them lived eight days; another five, and others from three to five days before dissolution took place; several died in strong paroxysms of pain, manifest by getting up and lying down very frequently, accompanied by violent efforts to urinate and defacate. The manure was usually covered with blood and mucus and the act of urination was very painful.

Mr. Garrison made three *post-mortem* examinations, and describes the following appearances:

> Lungs filled with air and infiltrated with mucus; the lining membrane of the air-tubes seemed slightly congested and irritated; the chest cavity contained some bloody-colored water; the contents of the paunch were hard, and the medicine which had been administered had not been absorbed; the contents of the manifolds in two of the animals was baked and dry as tobacco, and the folds softened and rotten; the intestines seemed swollen, and the lining membrane coated with mucus; the kidneys were of a greenish color, softened, and enlarged; the urine bladder was distended with dark-colored urine, one of them containing fully one gallon of clotted black blood; the heart was pale and flaccid; the blood in all three cases was darker and thicker than natural; it seemed to be more like paint than blood; the spleen was greatly enlarged, and the inside of it broken up into a pulp; the liver was thickened and filled with blood, the outside color being a glossy green, some parts more highly colored than others; the gall-bladder contained in one case one quart of thick, viscid, yellowish granular bile; the large blood-vessels along the spine seemed diseased, presenting an unhealthy internal surface; in one of the animals one of the horns became loose before death.

Loss, 11 out of 18; value, $277; 3 recoveries.

Joseph Sharp, living in El Dorado, said that he had 3 cows with their calves, and 2 weaned calves in Mr. Robeson's dry herd; they were put in on the 12th of May, and removed on the 25th of September. One of them manifested signs of sickness on the evening when she was taken home, and in a week thereafter she died. This cow seemed to suffer more pain during the middle of the day than in the morning or evening. At the time the first one died, two others were found to be sick; they both died; the last death took place on the 5th of October. Three of the calves were sick, but recovered. Loss, 3 out of 6; value, $125. *Post-mortem* examinations of 2 of the cows were made by Mr. Sharp. He found the bladder distended with a brownish-red urine, spleen twice or three times the normal size and very dark colored on the surface. In one of them, which had been purged by feeding corn and millet, the contents of the third stomach were found soft, in the other, the contents were hard and dry, "could be shaved down into chips," and the folds black and rotten. The blood in all 3 of them that died was too thick. Only 1 of these cattle passed blood with the feces.

Mrs. Smith lost one heifer in the Robeson herd on the 22d of September; another one, which she took home on the 25th, died on the 28th. She had only 2 head of cattle in the herd—value, $60. On the 23d of September Mr. Robeson called upon S. P. Barnes, a butcher in El Dorado, to make examination of 2 head of cattle that died in the herd. Mr. Barnes gave the following statement:

> I found one cow had been purging, and in this the contents of the third stomach were natural; the second cow had been constipated, and in her I found the contents

of the third stomach harder and drier than natural. The livers and kidneys in both animals appeared pale and faded in color, as if the coloring matter had been removed. In one I found the urine bladder was empty; in the other it was filled with bloody-colored urine; in the latter the inside of the bladder seemed hard and tanned, and almost black in color. The spleens were three times as large as natural, but the livers were not enlarged. One of the animals—a three-year-old cow—was not quite dead when I arrived at the place where she lay. I cut her throat, but hardly any blood escaped; the small amount which flowed was too thin and watery. The cow that I found dead, also, was almost destitute of blood. The tallow in both these cows was much too yellow, and I found this same yellow condition of the tallow in 8 or 10 other cattle which died afterward on the same range with the same disease. The meat of these cattle was light colored, like veal, and a disagreeable odor was present in all of the animals, dead and living. I never before saw any cattle sick with or die of Texas fever, but the moment I saw these I was satisfied that they died with that disease.

E. B. Cook, residing 7 miles northwest of El Dorado, stated that he placed 6 head of cattle—all cows and heifers—in the dry herd of Mr. Robeson on the 6th day of May, and took them away on the 23d of September. One of them died on the 27th, another on the 30th, and a third one on the 4th day of October. Two of them passed bloody urine. Two that were sick recovered. When he took them home he placed them into a field with 14 head of other cattle. None died but those exposed on the Robeson range. Loss, 3 out of 6; value, $100; 2 recoveries.

Mossman Bros. reside 8 miles northwest of El Dorado. History by Henry Mossman: On the 6th day of May 18 head of cows and calves were taken into the Robeson herd, and remained until the 25th of July. They were then sold to Bearsley & McAnaly. Twenty three head of steers were placed into this same herd on the 25th of July, and 17 cows on the 21st of September. On the 23d of September the steers and cows were taken home. One of the steers was sick on the day he was removed from the herd, and died on the day following; 6 more died in the course of three weeks, and 4 that were sick recovered. Of the 17 cows which remained in the herd only 2 days, 4 sickened, and 2 of them died—the first one on the 10th of October, the other one a few days later. The medication resorted to consisted in giving one gallon of melted lard at a single dose. In three cases this dose was repeated, and in a fourth one an addition of ten drops of croton oil was given. Out of the 4 so treated 1 died. None that recovered had been seen to pass bloody urine, but in a few the manure was covered with coatings of mucus and blood clots. Mossman Brothers made two *post-mortem* examinations, which revealed the following pathological conditions: Blood was contained in the bladder; in one blood extravasations were found in the region of the kidneys; also on the surface of the body, extending along the spine for a space of 18 inches. This one died twenty-four hours after the first evidence of sickness was discovered. The spleens were greatly enlarged; livers appeared about natural. The contents of the third stomach were normal in one case; in the other they were dry. Loss, 7 out of 23 steers, valued at $315, and 2 cows out of 17, valued at $60; 6 recoveries.

I tested the temperature of 4 of the animals that had been sick, which registered as follows: 103.2°, 103°, 102.6°, 102°; of one which had not been sick, 102.2° F. These cattle are all looking well, and are fattening rapidly. They are confined in a straw yard, and get all the corn they can eat. The hogs at Mossman's ate one of the dead cattle; ten days thereafter 4 of them were noticed to be sick; 3 recovered in about ten days; 1 died in five or six days. The one that died had a fit of apoplexy two months previous, and had never entirely recovered from the effects of it; was subject to involuntary discharge of feces and urine, and had an imperfect control over its movements. Matthew Robeson, a herder of cattle residing in El Dorado, made the following statement to me: That he has held a range for four successive years, extending from the corporate limits of the city of El Dorado 5 miles northwest, by 2 or 3 miles wide; that he has been herding cattle belonging to himself and to other parties on this range this season, beginning to take them in on the 17th day of April. The first death among his herd occurred on the 10th day of September—a cow owned by Mr. Saxton; the second one which died was an animal belonging to Mrs. Smith; she died on the 22d; on the day following 2 head died belonging to Mr. King.

He then had an examination instituted, and as a result of that examination notified all the owners of cattle in his possession to remove them forthwith. Mr. Robeson also stated that he received into his herd on the 20th day of July 75 head of cattle belonging to Davis & Connelly, butchers, of El Dorado; that some of these cattle remained on the range until the 28th of September. They were half-breeds with the exception of 7 head, which were supposed to be thorough Texans. The Texans were in poor condition when they were brought on the range, and were marked on the middle of the left side with an inverted A (∀) brand. Again Mr. Robeson stated that he received from Isaac King 300 head of cattle on the 9th of June.

Mr. King bought, during the winter, along the north line of Arkansas and south line of Missouri 1,000 head of cattle, collected them at Fort Scott in March, shipped them at the latter place, and unloaded at El Dorado on the 25th of April. He drove them on the range north and northwest of El Dorado, and there herded them until the 9th of June. He then drove 700 of them into Colorado on his cattle ranch, and placed the remaining 300 head under the care of Mr. Robeson, who already occupied the range at that time with a number of the town and other cattle. On the 9th of August 120 head more of the King cattle were taken into Colorado. I next saw Mr. Connelly, of the firm of Davis & Connelly, and got from him the following history:

> I bought on the 18th of July 68 head of half-breed cattle from Mr. Page. These cattle had been wintered on Deer Creek, 12 miles southeast of Caldwell, Sumner County, Kansas. When I bought them they were on a range, owned by Mr. Cox, 6 miles southeast of Caldwell, on Bluff Creek. I bought 7 head more to fill out the car; these were brought to Caldwell early in the spring from Benton County, Arkansas. I unloaded at El Dorado on the 20th of July, and placed my cattle in the care of Mr. Robeson. When I bought the 7 head of Arkansas cattle they were with a herd of 50 or 60 good grade native cattle, a $500 bull and several polled Angus cattle. None of the latter died during this season.

Mr. Connelly also informed me that Mr. Carter, whose farm adjoins that of Mr. Cox, lost 38 out of 50 or 60 head of cattle. Five of them were full blood polled Angus, and another a Hereford bull, for which Mr. Carter paid $1,000 last spring. All of Carter's cattle were held securely in an inclosed field, and have not been exposed to any foreign cattle.

On the 10th I saw Mr. James Mossman, who had just returned from Caldwell, where he had been to ascertain where the Connelly cattle came from. As near as he could discover the 68 head were bought from Mr. Carter; had been wintered cattle, half-breeds; that Mr. Carter lost many cattle at about the same time that the cattle began to die at El Dorado, and that Mr. Connelly had bought the 7 head from a Mr. Smythe (the agent for Mr. Donaldson, of Arkansas City). These 7 head were supposed to be through cattle from the Pan Handle, and were bought out of a herd on the Johnson and Hosmer range, near Caldwell, in the Indian Territory. On Sunday, November 11, accompanied by Dr. Bassett and Mr. Davis, I drove out to ex Brig. Gen. G. T. Wilde's, who, with his copartner, Colonel Mason, are engaged in the breeding and feeding of cattle. Colonel Mason was absent from home, but General Wildes gave me the following history:

> Mr. Mason visited Halstead, Harvey County, Kansas, in the month of August; here he found that a number of cattle had died—town cows—with the Texas fever, attributed to infection received through a herd of cattle brought from the line of Arkansas and Missouri; that these were the only foreign cattle brought into Halstead this season. These cattle must come very nearly, if not quite, from the same section of country as that from which the King cattle are said to be derived.

On the 12th I went out over the range where Robeson had herded his cattle during the summer. I found it to be an elevated ridge of limestone soil, extending for miles northward along the west side of the Walnut Creek. The Walnut Creek supplied the cattle with water morning and evening. The water was perfectly sweet and pure. At noon the cattle drank from a spring creek, which takes its origin from several springs located on the range. The grasses are the usual prairie grasses found throughout the middle section of the State, composed chiefly of bunch grass or blue top, blue grass, and here and there is to be seen small patches of buffalo grass, the latter occurring where there is alkali soil or the remains of what are called buffalo wallows.

I also went out with Mr. Robeson to examine and to see the condition of the King cattle. These are the only cattle which yet remain in the charge of Mr. Robeson and are located in a corral 2 miles west of town for winter feeding. I found them to be very diminutive cattle (mullet heads) and also in a very poor condition to withstand the inclemency of winter. Many of them were three year-old steers, but none of them exceeded 600 pounds weight. Many of them, however, have been sick, but have so far recovered as to begin to lay on flesh again. I tested the temperature of 7 head that had been sick, which registered as follows: 100°, 100.2°, 99°, 101°, 101.4°, 102°, 101°; of four that have not been sick, 97.7°, 101.4°, 101.3°, and 100.8° F.

Through the kindness of Mr. Robeson I am enabled to make an accurate tabulated statement of the number of cattle that were placed in his care during the season, the dates at which he received them, and the time when they were removed from the infected range:

Date of reception.	Owners.	Number received.	Number died.	Number recovered.	Value.	When removed.
April 17	Rev. S. F. C. Garrison	18	11	3	$277	Sept. 24.
17	Mr. Corey	5	1		40	Sept. 23.
17	Dr. Gordon	1				do
17	H. Saxton	1	1		40	
17	V. Brown	1	1		40	
25	Mr. Balch	3				Sept. 23.
May 2	Mr. Cupples	2	1	1	40	do
2	Mr. Collins	1				do
3	Mr. White	1	1		40	
5	Mr. Pascal	1				Sept. 23.
6	Mr. Carpenter	5	1	1	40	do
6	Mossman Bros	18				July 25.
6	E. B. Cook	6	3	3	100	Sept. 23.
6	M. Robeson	3				do
6	Mr. Jones	3	1		40	do
6	Mr. McAnaly	2	1		40	do
6	Mr. Stiler	1				do
12	Joseph Sharp	6	3	3	125	Sept. 25.
12	Mr. McIntyre	1				Sept. 23.
July 29	Mr. Glaze	3				do
Aug. 8	Mr. Sappington	1				do
23	Beardaly & McAnaly	100				Aug. 27.

Date of reception.	Owners.	Number received.	Number died.	Number recovered.	Value.	When removed.
June 9	Isaac King	800	26	25	$500	Ang. and Nov. 10.
9	Beardaly & McAnaly	3	1	....	40	Sept. 24.
July 20	Davis & Connelly	75	(*)	(*)	(*)	(*)
May 6	Mr. Tuttle	3	3	....	115	....
6	Mrs. Smith	2	2	....	60	....
July 25	Mossman Bros	23	7	4	815	Sept. 23.
Sept. 21	... do	17	2	2	60	....do ...
	Milk herd	124	9	....	325	....do ...
	Total	730	75	41	2,237	

* Were removed in small lots, September 28.

The milk herd contained exclusively milch cows from town, but were herded on the same range with the dry herd, although the two herds were kept separated for convenience. Mr. N. Rittenhouse herded 300 head of cattle north of the Robeson range, but he came no nearer than 1 mile to the range of the latter; neither have the cattle in the care of Mr. Rittenhouse crossed any of the trails of the Robeson cattle or been exposed to them or any other cattle in any manner whatsoever. They have been supplied with water from Walnut Creek, and have grazed over the same character of soil and grasses as have the cattle of Robeson's. Rittenhouse has not lost a single animal out of his herd during the whole grazing season.

In view of all the facts and the circumstances, as they appear to me, I am compelled to attribute the appearance of Southern cattle fever at El Dorado to the importation of Southern cattle by Mr. Connelly.

APPEARANCE OF DISEASE THREE DAYS AFTER EXPOSURE.

On my way to Harper, in October, I met Mr. E. F. Osborn, of Mulvane, Sedgwick County, Kansas, who related to me his experience with southern cattle fever, viz:

> On or about the 1st of October, 1869, my partner and I started from Salina with 240 head of high grade native cattle, bred by ourselves. We drove south 80 miles to Sedgwick City; then we crossed the through cattle trail, driving our cattle as fast as possible. We then drove 20 miles farther south and stopped for winter feeding. On the third day after crossing we found one high-grade cow sick; on the 5th she died, and a hundred more were sick; in ten days after the disease first appeared 200 of the 240 were dead. We then drove the remaining 40 head into the Arkansas River, and left them standing there in the water. Some of them were so sick that when they first went into the river they could hardly keep on their feet. Many of them remained there from seven to ten or twelve days; could not be driven away from the water; but they all recovered. These 40 head were the poorest in the whole herd of 240 when we started with them. Before we left Salina we had a hard frost, and during the drive south we had several frosts and continuous cool weather. We considered ourselves safe in starting after the frost; but to obviate all danger we endeavored to keep a safe distance away from the through trail; therefore we kept 20 miles west of it until we had to cross it; we then turned a square corner and drove across the trail.

I asked Mr. Osborn if it might not be possible that they crossed trails of Southern cattle between Salina and Sedgwick, but he said he was sure they had not. If their cattle were exposed only to the through trail then the stage of incubation in the first cow was probably less than three days, positively not more than four.

Respectfully submitted.

M. R. TRUMBOWER, V. S.

STERLING, ILL., *December* 20, 1883.

INVESTIGATION OF SOUTHERN CATTLE FEVER.

REPORT OF DR. H. J. DETMERS.

Hon. GEORGE B. LORING,
Commisioner of Agriculture:

SIR: In the following I have the honor of submitting my report on southern cattle fever. In my last report I took the liberty of stating what has been accomplished, and what yet remains to be done; also what I considered as the chief object of my investigation, namely, to discover the true cause of that apparently mysterious disease. My observations and experience of last year more than ever convinced me that the (exciting) cause of southern cattle fever consists in something intimately connected with or dependent upon the peculiarities of the Southern flora, and not—at least not directly—due to the climate and higher temperature of the Southern States, which only indirectly exerts its influence or aids in its production and propagation. The cause of southern cattle fever consists in something that requires for its production certain conditions given in the Southern States, or in those parts of our extensive territory in which the fever has its origin or permanent source. Some, perhaps most, of the conditions favorable to its development, and some of those unfavorable to the same, are known. If one carefully studies the facts communicated in my last report, he will find that decaying vegetable substances, a certain degree of warmth and moisture, and a low elevation above the ocean, are necessary requisites and important factors in the development of the infectious principle; while a low temperature, a high altitude, and, without hardly any doubt, an absence of moist and decaying vegetable substances are detrimental to its propagation. In proof of this, allow me to briefly restate some of the more salient facts, apparently in part contradictory of each other, but facts notwithstanding. As, however, my experience has only been with Texas and Western cattle I will limit my remarks to them, without intimating, though, that I regard the disease in question as an exclusive product of Texas or of the Southwest, for it is a well-known fact that other Southern States and the West Indies are just as well a source of southern fever as Texas.

1. Native Texas cattle never contract southern cattle fever, and possess immunity against infection as long as they remain on their native range or north of the same, provided they are not kept long enough north (in any of the Northern States) to become there acclimated, or, in other words, have never passed a winter in the North. But the same cattle if taken from their native range and driven or shipped south will gradually lose their immunity in proportion to the distance they go further south, and thus, if going far south, finally become liable to be infected and to contract the fever. This shows the infectious principle must be the more intense the further south the locality.

2. If Texas or other Southern cattle, to all appearances themselves perfectly healthy, are shipped or driven North, away from their native

range, after new grass has appeared and become interwoven or intermixed with the old dead grass of last year's growth, which, owing to the warmer weather and the usually abundant rains of the early Southern spring, is in a decaying condition, and these cattle, thus compelled to eat both the intermingled old and new grass, have but once taken a good meal of this mixed herbage, they will as soon as they arrive at a certain latitude further north infect every trail and pasture on which they graze, and every water-hole out of which they drink, with the infectious principle of southern cattle fever. And the native Northern cattle following them will, after some interval of time (period of incubation), contract the disease, as a rule, in its most fatal form.

3. If Texas or other Southern cattle are moved to the North before any new grass has made its appearance on their native range, or rather before the dead grass of last year's growth has commenced to decay, no infection of Northern pasture, &c., will take place, no matter how far north the Southern cattle may be shipped or driven. If, however, the cattle, thus leaving their native range in the South early in the season, or in the winter, should travel slow enough to be yet within a part of the South in which the southern cattle fever has its permanent source, when warm weather and abundant spring rains cause a decay of the old grass and start a vigorous growth of the new, the effect will be precisely the same as if the cattle had been kept that long on their native range; only the infectious principle imparted to the Northern pastures, &c., may be a trifle less virulent, and taken up by Northern cattle may cause a somewhat milder, though in a majority of cases yet fatal, attack of the disease. I had repeated occasions to observe that the fever, as a rule, is the more severe the further south the source of the infectious principle.

4. Northern cattle shipped to Texas, or to other parts of the South, will contract the disease, and as a rule die of it, if only once pastured soon after their arrival on land that contains both old and new grass—particularly if it is so-called hog-wallow land—or if only once allowed to drink out of a water-hole receiving the drainage of such land.

5. Grown Northern cattle imported into Texas usually contract the disease with more certainty, and in a more fatal form, than imported, Northern calves and yearlings. Whether such is the case because the latter have a smaller mouth, are more dainty eaters, and better able to pick out the blades of grass they want, and to refuse what they do not like, or whether their young organism is better adapted to resist the influence of the pathogenic principle, I will not now decide, and will only mention that some young animals, even calves, contract the disease in just as acute and severe a form as full-grown cattle.

6. In the North—say north of the southern boundary line of Kansas—the disease is only communicated through trails, pastures, and grazing grounds, or rather their grasses and other food-plants, and water holes previously infected by Southern cattle; but it usually does not make its appearance until the latter part of July or in August, or until the Northern prairies, fields, and pastures, owing to the heat and often abundant rains of the summer, contain a comparatively large amount of vegetable débris or decaying vegetation, which, it seems, is an important factor in propagating the pathogenic principle if once deposited. That a propagation of the once deposited pathogenic principle actually takes place on the grass or herbage of the trails, pastures, or grounds, &c., and outside of the animal organism, is demonstrated by the fact that the period of incubation, as a rule, is a long one, if the native Northern cattle immediately, or within a few days, follow the Southerners on the

trails, pastures, &c.; while it usually is considerably shortened if a few or several weeks intervene between the time at which the Southern cattle left and the time at which the Northern cattle entered the infected premises. As, however, the infectious principle is not volatile, and is not disseminated through the air or by winds, its propagation on the grass and herbage of the infested grounds may not be the sole cause of shortening the period of incubation, and the difference just stated may also, to a certain extent, be accounted for by the following fact: In about two, three, or four weeks after a herd of cattle has left its grazing grounds (trail, pasture, prairie, &c., as the case may be) a fine crop of young and juicy grass will be found, if the season is not unfavorable to its growth, wherever the cattle have grazed; while at all those places or spots where they have not been grazing the grass will be comparatively old and tough. If a herd of native or Northern cattle immediately follows a herd of Texas or other Southern cattle, which have infected the premises with the pathogenic principle of southern cattle fever, the former will principally graze where they find grass, and not where the Southern cattle have cropped it, and where they, at the same time, have deposited, as I shall explain further on, the infectious principle. But if the herd of Northern cattle enters the pastures, &c., formerly occupied by the Southern cattle two, three, or four weeks after the latter left them, or after a new crop of young grass has made its appearance, the former, for obvious reasons, will prefer to graze at the very places where the Southern cattle have grazed, and deposited the pathogenic principle. As it is well known that the length of the period of incubation depends, to a certain extent at least, upon the quantity and intensity of the infectious principle taken up by the animal organism, no further explanation will be necessary.

7. In higher altitudes, such as in Colorado for instance, the southern cattle fever, although sometimes appearing after an infection of the grounds by Southern cattle, is much less malignant than in the lower countries farther east, and comparatively seldom proves fatal, a fact which may find its explanation that in a higher altitude the atmosphere is thinner and dryer, and less charged with organic substances; besides, the temperature, on an average, is lower. All this is less favorable to a decay of vegetable substances and a propagation of bacteritic growth than the warmer climate and the more dense and moist atmosphere of a lower country. The dead or dried grasses of the Colorado plains, under the influence of the dry air, and often prevailing dry winds, are ground to dust, and thus disappear before any decay sets in.

8. The morbidly affected tissues of animals affected with or killed by the southern cattle fever, even if examined at once, invariably contain bacteria* of the micrococcus and bacillus kind, and it appears to be very probable, particularly in the light of recent research in regard to infectious diseases and their causes, that at least one of these two kinds of bacteria bears some causal connection to the morbid process. My own observations, examinations, experiments, and a careful consideration of undeniable facts, point toward the bacilli, and not to the micrococci. My reasons I shall take the liberty to state further on.

9. If all the facts known in regard to the communication of southern cattle fever to Northern cattle by means of trails, grazing grounds, pastures, water-holes, &c., are duly considered as they present themselves, there can hardly remain any doubt that the infection of the trails, past-

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* The word "bacteria," unless otherwise stated, is used as a generic term, because better understood by the average reader than Schizophytes or Schizomycètes.

ures, &c., must be effected by means of the saliva or slaver of the southern cattle. In proof of this assertion I may be allowed to state a few facts bearing on this point, and also to briefly dwell upon other theories now and then advanced. First, as to the latter. One theory charges the infection to a deposit of the urine of the Southern cattle. If it were the urine that causes the infection only those comparatively small and far apart spots in which the urine of the Southern cattle is deposited would be able to communicate the disease to Northern cattle, for it has been established beyond a doubt that the infectious principle is not carried through the air or disseminated by winds, and that even a wire fence separating a pasture occupied by Northern cattle from a trail or pasture of Texas cattle (*cf.* my last report) is ample protection. Besides, cattle are not apt to graze where another animal has urinated; and as the urine is soon absorbed by the ground or evaporated it could never be explained how it can be possible that the infectiousness of a pasture or trail increases in intensity, at least for several weeks after the Southern cattle have left it. If the urine constituted the vehicle of the infectious principle, the wholesale infection of every Northern herd of cattle that passes over and grazes on a trail of the Southerners, or feeds on a pasture that has been occupied by the latter, would hardly be possible, and, at the utmost, only one or a few animals of a herd would contract the disease. Another theory charges the excrements of Southern cattle with constituting the vehicle of the pathogenic principle. The objections just made against the urine theory will also dispose of the dung theory; besides, all cattle, but particularly grown animals, carefully avoid to graze where other cattle have deposited their excrements. They are apt to sniff at places where horses have voided their dung, and when suffering from certain digestive disorders, attended with a vitiated appetite, may even eat some horse manure, but they will never graze if they can help it where the dung of their own kind has been deposited, a fact well known to every cattleman. It may be possible that some pathogenic bacteria pass off with the dung, or even with the urine; but if they do, they most assuredly do not furnish the principal source of infection. Another theory charges the hoofs of the Southern cattle with being the communicators of the infectious principle. This theory, too, can be easily disposed of, even if it were possible that the hoofs were able to take up the pathogenic principle (bacteria, for instance), at the native range, and convey it to some other place, that other place could only be in the immediate neighborhood, because at every step in the grass the hoofs are wiped, and in mud or water they are apt to lose whatever may cling to them; besides, neither the horn of the hoof nor the skin of the foot constitutes the soil or medium needed for the reproduction, preservation, and propagation of such a pathogenic principle as that which causes the southern cattle fever. Even if the skin of the foot, particularly in the cleft between the hoofs, constituted a suitable medium, and afforded all the conditions necessary to the existence and reproduction of the pathogenic principle, the constant wiping and friction which those parts are subjected to on the march would preclude the possibility of conveying the principle (bacteria) in that way a thousand miles or even farther. Still another theory, which has yet a great many adherents even among practical cattle-men, charges the ticks often found on Texas cattle with being the bearers of the infectious principle, or even with constituting themselves the pathogenic agency. The principal objection that can be brought to bear against this theory is the fact that Southern cattle free from ticks will infect Northern pastures, &c., just as soon as those that have them, and that ticks of the same kind also occur in countries in which the southern cattle fever never originates or makes its appearance, unless it is introduced by Southern cattle infecting a trail, pasture, water-hole, &c. The perspiration (through the skin) of Southern cattle, and even the expirations (from the lungs) have been accused of constituting the pathogenic principle, or the vehicle of the same. But this theory, too, is fallacious, for, if true, the pathogenic agency would be of a volatile nature, and be communicated through the air, which it evidently is not, as already stated. Hence, the only thing that remains as the probable vehicle and medium of the pathogenic principle is the saliva or slaver of the Southern cattle deposited by them, not only wherever they graze and wherever they drink, but also often dropping in strings from their mouths when on their march. Any one familiar with droves of Texas and Cherokee cattle will have observed that they produce more saliva and slaver more profusely than any other cattle not driven, or at rest; and cattle, when grazing, while grasping with their tongues a bunch of grass, and drawing it into their mouth to be cut off by their incisors, necessarily soil the stubbles which remain standing with their saliva, particularly if slavering, as traveling Texas cattle always do. This saliva or slaver is somewhat sticky, and the microscopic organisms (bacteria) it may contain are thus temporarily glued to the grass that remains on the ground. The bacteria, thus deposited with the slaver (saliva and mucous secretions of the mouth), find a new soil which offers them all the conditions necessary to their existence and propagation, particularly if old and decaying grass or vegetation, as is usually the case, is existing among or between the stubbles of the grass that has been torn off. Dew and rain afterward provide the necessary moisture and also the means of further distribution. If the Southern cattle, before being shipped or started on their journey toward the North, take up on their native range or at any place between their Southern home and their Northern destination, but south of a certain latitude, the pathogenic bacteria of southern cattle fever—and there can be hardly any doubt that bacteria which have their source or origin in the South constitute the infectious principle or the cause of that disease—either with their food or their water for drinking, the bacteria, of course, will first pass into the paunch, where they find all the conditions (a suitable medium, warmth, and moisture) necessary to their existence and propagation. Ascending to the cavity of the mouth with the juices of the paunch when the animal is ruminating, they find a new and, at the same time, excellent medium in the saliva and mucous secretions, and thus it becomes possible not only that the bacteria retain their vitality, and that the same vastly increase in numbers, even if the journey of the cattle, as to time and distance, is a long one, but also that one herd of Southern cattle is able to infect a large territory (trails, pasture-grounds, &c.), at a long distance, a thousand miles or more from their native range. I might advance several more arguments in proof of the assertion that grazing grounds, trails, pastures, yards, water-holes, &c., are infected by means of the slaver, and that all other theories are untenable, but to do so will be in time, and can be done with much more force, after it has been proved beyond a doubt that a certain kind of bacteria constitutes the true and the sole cause of the disease. To conclude, I may be allowed to remark that all the phenomena of an infection—the non-volatile character of the infectious principle, the varying period of incubation, the more frequent occurrence o the disease in different seasons according to latitude, the killing of the infectious principle by continuous cold weather, or by a heavy frost, and the wholesale infection of Northern herds of cattle—will find a full explanation, if the slaver constitutes the medium, in which the pathogenic principle lives and propagates in the Southern cattle when taken north, and in which it is deposited on the grass, in the water, &c., while the same cannot be explained, if not the saliva, or rather the saliva and mucous secretion combined, but something else constitutes the medium.

As above stated, I have reasons to believe that of those bacteria found in the morbidly affected parts, particularly in the liver and in the spleen of cattle affected with the southern fever, the bacilli and not the micrococci constitute the pathogenic principle, or bear a casual connection to the morbid process. Still, I will not deny that the micrococci, too, may possibly possess septic properties, particularly if obtained from a part in a state of dissolution, for instance, from the spleen, an organ which I invariably, at every *post-mortem* examination, found to be in a disorganized condition, even if the affected animal has been killed by bleeding or by a pistol-ball. Such micrococci, if inoculated into the organism of a healthy animal, may have a septic effect, and may even cause disease and death, and still may not constitute the infectious principle of the southern fever. According to what is known of the behavior and the pathogenic action of the various known pathogenic bacteria the morbid process and the morbid changes in southern cattle fever point toward bacilli and not at all toward micrococci as the probable cause. Particularly the fact that the infectious principle, whatever it may be, is never conveyed through the air from one place to another, and requires in order to produce morbid changes in an animal a very long period of incubation, and then rather suddenly develops its malignant action, it seems to me almost excludes the possibility of a micrococcus constituting the cause. It is true, in anthrax, a disease known to be caused by a bacillus, the period of incubation is a very short one, at least in those cases in which the disease is communicated from a diseased to a healthy animal, but the attack invariably is a sudden one, and *Bacillus anthracis* not only shows a very rapid propagation, but is also otherwise entirely different from the bacilli found in southern cattle fever. A micrococcus, as a rule, propagates too rapidly to require a very long time for the development of its pathogenic action, and would fill the whole organism, and very likely be found in every drop of blood, long before the sometimes very long periods of incubation of the southern fever has expired; besides that, every infectious principle known to consist of micrococci or diplococci is more or less volatile and can be communicated through the air, while those consisting of bacilli usually show a different behavior. But of course, if there were no other reasons, those just given, resting only upon analogy, might not carry much weight, or decide anything, and might be met by saying that the micrococci or diplococci found in southern cattle fever may be entirely different in their behavior from any other known species of pathogenic micrococci. There are, however, some other facts which tend to show that the bacilli and not the micrococci most likely constitute the pathogenic principle.

1. The bacilli are a constant occurrence in the diseased parts, but particularly in the liver and in the spleen of cattle that are affected with or have died of southern cattle fever.

2. The bacilli sufficiently differ in shape and size from all other well-known species to be at one recognized when seen under a sufficiently high power, a fact which I intend to demonstrate, not by a description or by drawing, but by photo-micrographs, as soon as I shall be able to

obtain fresh material in which the bacilli have not been subjected to any change whatever by the action of hardening fluids or reagents.

3. In three sections of liver and spleen recently mounted in balsam, but cut last winter from pieces of liver and spleen which were hardened thirteen months ago, when perfectly fresh, in alcohol and in a a solution of bichromate of potash, and have since been preserved in alcohol, the bacilli are yet intact, and under a high-power homogeneous immersion objective are easily recognized as the same kind of bacilli which I never failed to find in the diseased livers and spleens when examined fresh. Besides, the bacilli do not merely adhere to the surface of the sections, but appear to be imbedded in the tissue, because they are found at different depths, and require to be seen under a high power and different focusing, according to the plane in which they lie. The sections, when cut last winter, were stained in "Beale's carmine," and before being mounted were restained in an aqueous solution of methyl-violet (1:500), but after they had been in alcohol and oil of cloves, and been mounted, the aniline staining had almost entirely disappeared, been washed out by the alcohol and oil of cloves, and thus the bacilli, which did not take the carmine stain, appear but indifferently stained with methyl violet, and are rather pale, but are plainly seen and easily recognized.

4. The micrococci or diplococci that may have been present in the liver and spleen are now absent, at least cannot be found notwithstanding a most careful search, which proves to me that the same, if they have been present in those tisssues, must have been there by accident, or have existed only in the fluids, and have never been imbedded in the solids like the bacilli. In my opinion the facts just related plainly show that the presence of the bacilli cannot be an accident, but must have some connection with the morbid process.

Last year, when investigating the southern cattle fever in the Southwest, and even before, when I made my first observations on that disease and several *post-mortem* examinations of cattle that had died in Champaign, I became fully convinced that I had to deal with a bacteritic disease, or with a disease that owes its existence to some pathogenic bacterium. As it is admitted that the southern cattle fever has its origin or permanent source in the South, I could not help arriving at the conclusion, after taking all the peculiarities presented into consideration, that the pathogenic principle (bacterium) must be connected with, or be dependent upon, the flora of the Southern States, and cannot be the direct product of the climate or the higher average temperature. Having found the bacilli in the morbidly affected tissues several years ago at my first examination of the southern cattle fever at Champaign, and considering them, even at that time, as the probable, or at least possible, cause of disease, I naturally looked, when in Texas, for something corresponding to be found on, or to be connected with, the herbage and grasses which constitute the food of the cattle on the Texas cattle ranches. The well known experiments of Dr. Buchner directed my attention to infusions—natural and artificial—of the dead and decaying grasses of Texas hog-wallow land—to those of the latter in particular, as it was repeatedly stated by experienced Texas ranchmen that hog-wallow land is considered as the most dangerous grazing ground for cattle recently imported from the North. The natural infusions I found in the spring prepared in the so-called hog-wallows themselves, and the artificial infusions were made by putting some of the dead grass in a vessel and pouring rain-water over it. When examining my infusions, I found, as could scarcely otherwise be expected, a variety of microscopic organisms (bacteria), but among them, in larger or smaller numbers, invariably a bacillus which, in every respect, closely resembled both in size and form those bacilli which I had found before and found afterwards in the liver and spleen of the diseased cattle—certainly something worthy of further investigation.

As I look upon it, the principal object in investigating an infectious disease, particularly if the same is very fatal, causes great losses, and is capable of spreading a great distance, must be to ascertain the true cause, and to become acquainted with its nature and its mode of action, and the means and conditions necessary to its existence, propagation, and communication. As long as we are in the dark in regard to the cause of a disease, particularly if the latter is infectious, our treatment and our prophylactic measures can only be of an empirical character, and at best be very uncertain. But as soon as we know the cause, its mode of action, its means of existence, and its manner of propagation and communication, we have gained a great advantage, for then, if the cause is accessible and can at all be destroyed its effects can be neutralized or its propagation and communication can be prevented. The possibility is then given to devise rational measures which will have the desired effect. Therefore, sincerely believing, nay, almost convinced, that my endeavors to discover the cause of the southern cattle fever are in the right direction, it is and has been my desire to subject my conclusions, above stated, to a practical test, and to decide by experiment whether the same are correct or erroneous. Last year I made some efforts in that direction, but the experiments, for reasons stated in my last report, did not meet with satisfactory results, and some unavoidable mistakes were committed. So, for instance, I inoculated a Texas cow, which undoubtedly possessed immunity, but was the only animal at my disposal, and as I had to remain in Texas I delegated that part of my experiments, of which success might have been expected, because to be carried out in the North, where the cattle do not possess immunity, to a friend. It also miscarried for reasons stated in my last report. Another experiment, made by myself in the North, was made on very young animals, and too late in the season, and besides this, some other mistakes, partly unavoidable, and known to be mistakes when made, and partly due to a want of facilities and inexperience, were committed, which it will not be necessary to enumerate, for they will be avoided in the future. All this combined, however, amply accounts for the want of success, which, therefore, has not decided anything. Besides it is to me exceedingly doubtful whether a disease not known to have ever been directly or indirectly communicated by a diseased animal to a healthy one can at all be inoculated in the usual way. At any rate no well authenticated case of any direct infection or communication of the disease from a diseased animal to a healthy one is on record, while many cases are known in which animals took sick with southern cattle fever and died of it in the midst of healthy herds and none of the heathy animals, unless previously infected, ever contracted the disease. Neither is it positively known that Northern cattle or cattle themselves susceptible to an infection ever infected northern pastures, &c. Still, whether under certain circumstances they are able to do so is another question. Northern or susceptible cattle, grazing on premises or drinking out of waterholes infected by Southern cattle, almost invariably contract the disease and die of it before the system becomes accustomed to the action of the pathogenic principle (the bacteria), and charged with the same to such an extent that the latter will be present, and be constantly reproduced in the mucous secretions of the mouth and in the juices of the paunch.

But it stands to reason, if they (the Northern cattle) gradually acquired immunity like the Texans, and then continued for a certain length of time to occupy infected premises and to take up the infectious principle, or if it were possible to charge their system with the pathogenic principle, as just indicated, before they contract the disease and die of it, then these Northern cattle, if driven to uninfected pastures, would probably infect the latter just as effectively and just as soon as Southern cattle, particularly if the Northern cattle were first driven like a herd of Texans, and thus caused to slaver. That susceptible cattle, or such as will contract the disease, do not communicate it to others, strange as it may seem, probably also admits an explanation, if all known facts are taken into due consideration.

In the first place, the pathogenic principle (the bacteria) of southern cattle fever does not seem to be indigenous to the animal system, but very likely belongs to the decaying grasses and herbage of the South, and only by what may be called accident enters the animal organism. In Southern cattle, or in such as possess immunity, that is, in such in which the pathogenic principle (the bacteria) produces no morbid changes important enough to seriously disturb the health of the animal, the organs, which constitute the principal seat of the morbid process in diseased cattle, the liver, spleen, &c., it seems, have either become accustomed to the action of the bacteria, or else have gradually become sterilized ground, and thus cease to be a favorable medium. That such is the case will be understood, if it is kept in mind—1, that the first introduction of the bacteria into the organism of Southern cattle takes place while the latter are young calves, which, as is well known, possess much less susceptibility than grown cattle; 2, that the number of bacteria taken up the first time undoubtedly is a comparatively small one and not sufficient to cause serious mischief, but just large enough, particularly if supplemented by successive small invasions, to gradually cause an immunity, which, although but temporary, will last for some time after the invasions have ceased. As long as Southern cattle occupy infected territory the bacteria will enter their organism with the food and water for drinking, and finding in the paunch all the elements necessary for their development and propagation, many of them probably reach the cavity of the mouth by ascending with the food and the juices of the paunch during the process of rumination, and then in the mucous secretions and saliva again find a favorable medium in which their existence and propagation are fully secured, and by which they become glued to the grass, &c., as has been above explained. It is possible that a great many of the bacteria taken up with food or drink, or developed in the paunch, and, may be, the majority of them, pass on with the food through the digestive canal, and are discharged with the dung; but if they are they will be comparatively harmless, because they will be confined to those spots at which the dung is dropped, and at which other cattle, as a rule, do not like to graze.

If Northern cattle, not at all accustomed to the action of these bacteria, take them up with this food or drink for the first time, the bacteria likewise enter the paunch, and propagate in that organ, but passing on into the other stomachs and the intestines, they probably cause increased activity and increased absorption, or even lesions, by irritating the mucous membrane, and thus may find their way into those organs—the liver and the spleen—in which afterward the morbid process of the Southern fever has its principal seat, while in Southern cattle such an irritation of the digestive canal, which in them has become accustomed to the presence of the bacteria, is either very limited or does not take place. That the bacteria, or whatever may constitute the infectious principle, produce irritation and congestion in the digestive canal, resulting in most cases in increased absorption in the third stomach, and in lesions in the fourth stomach and a part of the intestines, is demonstrated by the morbid changes usually found in those organs at *post mortem* examinations. It is, however, also possible that the bacteria, or a large number of them, reach the liver, the principal and, according to my observations, primary seat of the morbid process, in a more direct way—through the duodenum. The bacteria, if once passed beyond the second stomach, or domiciled in interior organs, for instance, in the liver, spleen, &c., cannot very well ascend to the mouth, there mingle with the saliva and mucous secretions, and thus be deposited on the ground; consequently Northern cattle cannot very well infect pastures, &c., unless every day a new lot of bacteria is taken up and propagated in the paunch, as is the case with Southern cattle. Of course the explanation just given I wish to be considered only as a strong hint. Something more definite may be said after it has been conclusively demonstrated what bacterium constitutes the true cause of the southern fever.

When returning to Texas last spring—I arrived in San Antonio about the 1st of May—it was my intention, as I explained on the 24th of April, when in Washington, to collect and to prepare what I believe to be infectious material, and to return to the North in about a month to complete my preparations (bacillus cultivations, &c.), and then to put them to a practical test. I intended to return to the North for two reasons: First, no southern cattle fever could be found in Texas, nor could it be expected to occur before December, because the native Texas cattle possess immunity, and the Texas ranchmen, at least the more intelligent ones, and all those who have had any experience with the Southern fever, take care not to import any Northern cattle except late in the fall and in the winter, for they have found that to be the least dangerous season of the year. Secondly, my proposed experiments could only be expected to give satisfactory results if made on susceptible Northern cattle, and at a place where a natural infection is out of the question. But in the latter part of May, when my preparations were nearly completed, and I about ready to leave for the North, circumstances beyond my control compelled me to remain in Texas. Of course my plans could not be carried out, my preparations could not be used, and the question I was so anxious to decide for the time being had to remain unsolved. I had orders to investigate, besides southern cattle fever, also other infectious diseases of more than local importance. But in Texas the infectious and contagious diseases of domesticated animals, which are of general interest, or of an epizootic character, are very few in number, and without any fear of contradiction I may say there is hardly a country on the globe in which cattle and live stock in general are less subject to disease than in Texas. It is true Texas ranchmen sometimes suffer great losses, particularly in the latter part of winter, but these losses are not caused by disease, and almost without exception result from want of food, want of water, or want of shelter in inclement weather. Losses that occur during the summer months are far less severe, and are mostly caused by insects and their larvæ.

Besides the southern cattle fever, which does not, at least not visibly, affect the native Texas cattle, the only infectious disease of any importance is so-called "black-leg" or "black-quarter" (the *anthrax symptomatique* of the French), but even this disease occurs only at certain localities and during certain seasons of the year, particularly in the spring, and in my opinion will become a rare occurrence in the grazing districts of Texas as soon as Teasx stockmen will learn that dead animals must be buried or be cremated, and that it is bad policy to allow live stock to drink the water of stagnant pools. The infectious and epizootic diseases occurring among sheep were investigated last year, and although I do not claim that everything worth knowing about them has been brought to light, enough is known to enable the Texas flock-master to apply such measures of prevention as will protect his flocks. Besides, the spring is not the time in which those sheep diseases make their appearance, and, owing to a severe winter (severe for Texas at least), hardly any case occurred or came to my knowledge. Last year an epizootic disease occurred among the horses on the Gulf coast, but this year nothing of that kind happened, at least not as far as I was able to learn. So not much could be done, except examining some cases of so-called "black-leg," and in regard to these I was not able to ascertain anything that is not already known, because being in a thinly settled country and far from home I lacked the necessary facilities, such as a laboratory and experimental station. If it is desired to study and to thoroughly investigate the disease known as "black-leg," it can be done more easily and with better facilities in a more thickly settled country, where the distances are not so great as they are in Texas. Hence toward the end of July, or about the first of August, I asked the Department, briefly stating my reasons, to be recalled from Texas, and in reply to my letter I received orders which assigned me to other work. So I left Texas in the forepart of August.

Very respectfully submitted.

H. J. DETMERS.

DECEMBER 1, 1883.

CONTAGIOUS ANIMAL DISEASES.

BY EZRA M. HUNT, M. D., SC. D., TRENTON, N. J.

The relations of domestic animals to the public health—to food and milk supply—the comparative study of their diseases as throwing light on human ailments, and the immense financial and commercial import of any serious diseases occurring to them, cannot but impress any one who will give to the subject that consideration which its importance demands.

In 1862 the medical officer of the privy council of Great Britain made an important report on the diseases of live stock in their relations to the public supplies of meat and milk. In that paper Prof. John Gamgee states the number of horned cattle in the United Kingdom at 7,646,998, and calculates the loss by deaths among these animals at £6,000,000. The census of 1880 states the number of food animals in the United States at 91,805,232. The chief epizootics named in the report referred to are rinderpest, or typhoid or enteric fever of cattle, which always spreads from the Russian steppes; contagious pleuro-pneumonia of cattle, a disease always extending from Central Europe, though probably traceable to Asia and Africa, in some parts of which it is a very common disease; the epizootic aphthæ, murrain, or the foot-and-mouth disease, and sheep-pox. Of the enzootic diseases, which depend on local causes, and one parallel to endemics in man, anthrax or carbuncular fever takes the lead. Of this there are so many varieties of classification and description that we cannot yet be said to have a settled nomenclature. Thus, splenic apoplexy, braxy in sheep, the black-leg or quarter-ill of Britain, and other erysipelatous forms in the sheep and pig, boils and carbuncles, parturition fevers, hog cholera, Texas cattle fever, and some other ailments have been included in this class. Add to these the parasitic diseases of animals, and we have a score or more of diseases which are either deadly to the animals or injurious to meat and milk as food products. Many of these are communicable, not only to different varieties of animals, but to human beings as well.

None of these diseases are claimed to have originated on American soil, except it be the Southern cattle fever, which is regarded by many as only a variety of anthrax. When we consider, too, that rinderpest, pleuro-pneumonia, and foot-and-mouth disease were brought to Great Britain from the Continent, and that the ravages of these diseases have cost and are costing the British Government millions of pounds sterling annually, is it not wise for us to accept the signals of precaution and prevent them from becoming indigenous? As yet rinderpest and foot-and-mouth disease have not obtained a foothold, and contagious pleuro-pneumonia has not passed the point of possible extinction. Pleuro-pneumonia reached England about 1842; foot-and-mouth disease in 1839; sheep-pox in 1847. Of rinderpest there have been four outbreaks, viz., in 1745, 1865–'66 (the most destructive one), 1872, and 1877. We have been singularly fortunate in that we have escaped two of these formidable diseases. Rinderpest is not very likely to obtain a foothold here, but foot-and-mouth disease, with its great contagiousness and its immense loss to milk-producing animals, is greatly to be feared. Fortunately, rumors of an outbreak in the Far West proved to be unfounded. But the fact is well known that more than once it has arrived at our ports from abroad, and has been prevented from spreading here by rigid inspections on arrival and close quarantine after landing. During the past year the disease has been so prevalent in the United Kingdom as to cause widespread alarm and great pecuniary losses.

We propose in this paper to state some facts, observations, and opinions as to contagious pleuro-pneumonia, and also, in connection with the detail of some experiments as to foot-and-mouth disease, to offer a few comments upon it.

CONTAGIOUS PLEURO-PNEUMONIA.

The general course and symptons of pleuro-pneumonia are so well known, and have been so fully and accurately described in the reports of the United States Department of Agriculture, that there is no need of repeating them here. The only points upon which perhaps there is need of more extended observation and a more concurrent testimony is whether climate or other conditions have modified this disease as found on American soil, and whether different grades of stock are equally subject to its ravages. No one can see much of the disease without being struck with the great variations in its malignancy. We have seen outbreaks in which every animal attacked seemed early to become mortally sick, and where one or both lungs changed in a short time from a weight of three pounds to over twenty. In other cases the course of the disease has seemed mild, and most of the animals were likely to recover. This has led to a distinction among some veterinarians, so that they have come to speak of certain cases as English or European, and of others as American cases. It would be a good service if this Department could obtain the comparative statistics of English and American cases, and the accurate testimony of creditable veterinarians who have had in charge many cases both in this country and abroad.

It has been asserted that in certain exposed sections, as on Staten Island, common pneumonia is frequent and fatal among cattle, and that some of these have been mistaken for contagious pleuro-pneumonia. We last year offered to visit, on notice by telegraph, any such cases of common pneumonia, which an extensive practitioner there asserted he had frequently seen, but as yet have received no such notification.

The contagious character of pleuro-pneumonia in this country has certainly not diminished with the change of climate. Still there is some reason to believe that our severer winters tend to freeze out the disease, and, where there has been a full exposure, give us a better hope of interrupting its contagiousness; yet the contagion itself seems very persistent unless there is subjection to freezing and to various methods of disinfection. It is well established that after an outbreak had ceased for a year or more, the removal of the old barns and the exposure of the unfrozen ground beneath at once revived the disease. A case not long since occurred on Long Island where the transfer to the new sheds and the destruction of the old, near by, seemed to originate or reproduce the plague. Facts are accumulating to show that the accidental protection from frost caused by shelter seems to continue some of the contagions both of men and of animals. This is one of the contagions probably not wafted far, but quite indestructible unless largely exposed to air, to severe cold, and to disinfection.

But the most important question of all pressing upon our attention at the present time is whether we are to forsake the method of stamping out the disease by occision, and to substitute the system of inoculation as first practiced in Belgium and Holland, and as revived in Scotland under the auspices of Rutherford and Williams, and as sanctioned also by Fleming and others. This is the more important because, under the advice and approval of the Drs. McLean, the board of health of the city of Brooklyn has allowed or authorized this system in its stables, and at a point where the disease has long had a foothold. At one time Professor Law expressed the hope that the inoculation method, under some modifications, would be revived. In New Jersey the State law permits inoculation, under expert oversight, in a herd in which there has been an outbreak, if such inoculation is ordered or approved by the board of health. In the last instance the law was based on the views of Rutherford, Williams, &c., and on the fact that the State despaired of securing riddance of the disease, with reasonable expenditure, if it must be constantly subjected thereto by the transportation or driving of cattle from infected localities in other States.

The details of inoculation as followed out in New Jersey are of much interest. Through the influence and practice of Mr. Lamerz, a German veterinarian of Newark, it has for several years been a custom with dairymen in Essex County, and especially about Orange, to inoculate their herds, not only when there was an outbreak among their cattle, but as a customary precaution. It is worthy of note that all who have pursued this plan express themselves fully satisfied therewith, and aver that they have never known any case of the transmission of the contagion by this means. While much of this is negative testimony, and may be partially set aside by saying that most of them would have escaped contagion without this, it is yet worthy of record that while there have been sporadic cases of the disease in Orange and adjacent localities, these have been no more numerous than at some other points. For over a year it has been the habit of the veterinary service of the State board of health of New Jersey to seek to eradicate the disease by slaughter if only one or two cases had occurred. But if there had been more extended seizures before notice, or if the disease threatened to spread, resort has been had to inoculation. Dr. J. W. Hawk, of Newark, and Dr. Leatherman, of Clinton, have had occasion thus to inoculate several herds. In about two hundred cases of inoculation they report that the results have been altogether satisfactory. Herds have been protected and the disease limited. While a few of the animals have lost their tails, and some have been for a little time quite sick, no deaths have occurred from this cause. Great care has been taken in the selection of the inoculating juice, and it has been generally introduced, by a seton of woolen yarn, into the muscle just beneath the skin of the tail.

While thus feeling our way on the basis of the authorities before alluded to, and by a cautious use of the method in general acceptance, the following series of cases occurred:

A farmer and dairyman having about thirty head of animals in his herd, and not having and never having had any cases of contagious pleuro-pneumonia among his cattle, was informed that two or three of his neighbors had the disease among their herds. He was a mile and a half distant from the nearest one. The cattle had not come near to each other by neighboring fields. He had no occasion to take any of his cattle to other yards, or to have any brought to his. He had not purchased an animal for three years. Not knowing the law of the

State, he concluded to have his cattle inoculated by a veterinarian of Newark. His entire herd was inoculated in the month of December. One or two of these animals lost their tails, several were somewhat sick and recovered, and did not contract pleuro-pneumonia, though afterward exposed. On the tenth day after the inoculation two calves had convulsions and died. Three of the healthiest cows were taken severely sick with all the symptoms of contagious pleuro-pneumonia, and had to be slaughtered. *Post-mortem* examination left no doubt as to its being genuine contagious pleuro-pneumonia. Some of the other cows did not seem to recover their usual health or milk supply. A thorough examination four weeks later by H. W. Rowland, D. V. S, of Jersey City, and J. Gerth, jr., D. V. S., of Newark, showed conditions of lung that would indicate nothing else than contagious pleuro-pneumonia. As the cows were gaining, the owner was permitted to keep them, on condition that they should not be sold until fit for slaughter, and then not for any other purpose.

Here we have a series of cases in which either the veterinarian caused the disease to the herd by person or fomites, or introduced it by inoculation. If the latter, it would seem to support the hypothesis of some, who, while admitting that the introduction of the virus into a muscle, instead of by the breath or into the lung, generally causes a milder disease, which is protective, yet that occasionally, under conditions not yet known, it will resume its malignant activity and light upon the organ which seems to afford its chosen nidus. It seems somewhat analogous to the old facts as to the inoculation for small-pox, in which undoubtedly the introduction of the virus into the skin or flesh instead of into the lung did modify and mitigate the disease. But in this it was a known fact that now and then a strange exception would occur, resulting in secondary fever and death. While single cases prove but little, and the mind must suspend its judgment until other cases occur, or until the occurrence is explained, it must be confessed that these cases were a restraint upon what had before seemed to us to be legitimate conclusions. The great asserted facts upon which the more recent hopes as to inoculation of cattle have been predicated are (*a*) that the virus thus introduced never causes any affection of the lung, and (*b*) that the animal that has been inoculated does not impart the disease to others.

In view of the immense interests involved, this Government should institute a series of experiments to settle this matter, and either arrive at the conclusion that systematic and rational extinction of the disease is to be secured by slaughter, or define how it can be aided or secured, if at all, by systems of regulated inoculation. Since the important experiments and deductions of Pasteur, Chauveau, Touissant, Koch, and many others, it seems almost imperative that this Government should, by systematized methods of histology, pathology, and laboratory investigations, and by the experience of skilled observers, determine the means of checking those marauding epizootics that imperil the health and life of so many millions of animals of various species, and with it imperil the industrial interests of all classes and the very life and health of a race so dependent on good meat and good milk for food.

FOOT-AND-MOUTH DISEASE.

We are fortunately dependent on foreign authorities for the most accurate descriptions of this disease. Unlike pleuro-pneumonia, it is readily communicable "to sheep, goats, swine, and poultry; it is easily transmitted to the human subject. It has been described as existing in the horse, dog, wild fowl, deer, wild boar, cat, &c." The milk of animals in many cases seems to have conveyed the disease to man, although some regard this as having occurred only when there were vesicles upon the udder or teats, the secretions from which had mingled with the milk. (Walley, Edinburgh, 1879.) The same author also speaks of it as "one of the most infectious and contagious maladies which affect domestic animals, and the easiest of transmission," and as remarkable "in the effect which the milk of animals affected with it produces on their young and even the young of other species." Because of its rapid and diffusive contagion, and of the fact that one attack does not protect from another, that both the meat and milk supply are jeopardized thereby, it is probable that its actual losses to food products are greater than that of any of these pervasive plagues. Recently J. W. Stickler, M. D., of Orange, N. J., a physician who had become interested in the disease only because of its comparative relations and its partial similarity to certain aphthous diseases of children, procured from Professor Williams, of Edinburgh, some of the virus of the disease, and inoculated some calves therewith. The saliva was taken from a cow affected with the foot-and-mouth disease, and put into glycerine tightly corked in a bottle, and immediately forwarded to the doctor. In a recent note to the writer, Dr. Stickler says:

> The history of this case is as follows: Alderney calf, two and one-half weeks old; before inoculation seemed perfectly well; visible mucous membranes free from any eruption and discharge; skin and interdigital spaces normal in appearance. I inoculated the animal by subcutaneous injection of the virus just posterior to the elbow joint and anterior to the stifle. Nothing at all positive developed till January 2, when the temperature rose to 104°.8 Fah. There was a discharge of quite tenacious mucus from the nostrils, and at various points upon the mucous membrane of each there were small papulæ. The mouth was hot and red, although there were no distinct ulcers or aphthæ. The nearest approach to an ulcer was an apparent thinning of the mucous membrane at one or two points. The saliva seemed to be increased somewhat in quantity. The bowels were loose. A microscopic examination of the blood showed the existence of small round or oval bodies of a faint port-wine color. They had an activity which, I think, was independent of that caused by any motion of the blood plasma. To determine this point, I was careful to place upon the glass slide only a drop of blood, adjusting the cover glass with sufficient firmness to cause an even dispersion of the fluid. I then noticed that these little bodies seemed to have the power to move in various directions. The saliva and nasal discharge contained the monads spoken of in the various works upon "cattle diseases." I am not aware, however, that attention has been called to the existence of small, active bodies in the blood of animals affected with "foot-and-mouth" disease. The feet now became slightly reddened, but showed no blebs or ulcerations. There was also a little swelling just above the hoofs. The condition of the calf remained essentially as just given till January 10, when the feet became swollen to a marked degree and the redness more pronounced. In the interdigital spaces the skin was found to be loosened from its attachment, although there was no fluid to be seen. There was but little heat of coronets. The back of the animal was arched. The redness of the feet became more intense, the hair coming off, leaving quite large areas of uncovered red integument. The bowels again became loose, the animal feeding insufficiently, till, finally, on January 17, it died.

As seen by us the disease was in its subacute stages. One calf had just died of it. When the vesicles break, the red or scarlet surface becomes covered with a secretion, but shows little tendency to return to its normal condition. The emaciation of animals is rapid, both from the disease itself and the interference with feeding, which it causes.

We do not know of other experiments with the virus in this country. It is very certain that the most scrupulous vigilance should be exercised as to the disease, and that it should never be allowed to obtain a foothold here. There is no disease against which the veterinary inspector in England watches with greater care. Our system of large herds and cattle ranches has no parallel in the cattle culture of the United Kingdom. Its spread here would, if it should get full headway, compare with that abroad as does the sweeping fire of the prairie with that of a city block, where the provisions for extinguishment are ready to the hand. Well may all owners of cattle urge the National Government to exercise its preventive discipline over importation and all movements of cattle from the seaboard, all the more because recently a new demand has sprung up in the West for young stock from the East. The transportation which has heretofore been toward the seaboard is now likely to be met by a counter-current to be inland west, since the stock-breeding there does not supply the demands which the fields of space in the middle land between the oceans provides for the feeding and fattening of young cattle.

We scarcely need to emphasize the application of the same precautions as to those diseases of swine and sheep which are equally destructive to these smaller flocks, and which alike imperil interests in which large capital and great commercial enterprises are involved. The census of 1880 gives as for the United States 10,357,488 horses, 1,812,808 mules and asses, 12,443,120 milch cows, 903,841 work oxen, 22,488,550 other cattle, 35,192,074 sheep, and 47,681,700 swine, or an aggregate of, or about, 131,000,000 in all. When we consider that the yearly increase is constant, we ought not to need extended argument to show that the sums expended in competent investigation and in skilled oversight of these interests, if honestly and intelligently expended, is among the very best investments the General Government can make. Perhaps the lack at present is more in competency of observation and in tried and successful methods of protection than in a recognition of the desirability of such oversight. But as a demand creates a supply we are already seeing Harvard University and the University of Pennsylvania with veterinary departments, Toronto and New York with worthy veterinary colleges, and the American Public Health Association giving it prominent consideration, the medical profession alive to its importance and co-operating here with something of the same spirit and ability with which in England the foremost member of the Royal College of Physicians and Surgeons is found conducting and aiding in investigations of a similar kind.

It is only by a combined and continued system of surveillance that we can hope to prevent or arrest the wandering epizootic pestilence or those enzootics which spring up in localities and are dependent upon causes which, although difficult of detection, are, in the light of the past few years, likely soon to be unraveled. By such a course, and by putting on record the facts and experience obtained by skilled local observers, we shall succeed in arresting or abating many of the vagrant diseases, and thus greatly appreciate both the wealth, the comfort, and the health of our people, and be able to furnish the markets of the Old World with a surplus meat supply excellent in quality and abundant in quantity.

GLANDERS AND FARCY.

The disease known as glanders or farcy is so insidious in its character as to need most careful inquiry and inspection on the part of local, State, and national authorities. Unless large powers are given, the laws will be evaded and the disease perpetuated. This has been signally illustrated in a series of cases that have occurred in the South Orange car stables, in Newark, during the last year. There is reason to believe that over a year since a case of glanders occurred in those stables, which was claimed to have been cured. About August 1, 1883, the attention of the local boards of South Orange and Newark, and of the State, was called thereto through a legal process of inquiry. The disease was found so extensive that about sixty head of horses had to be killed. Not long after the veterinarian in charge, Dr. J. W. Hawk, of Newark, condemned three more. The owner consented at once to their destruction. There was delay as to one, which had the least external manifestation, and during the delay the owner, under unfortunate rival advice, concluded that the animal was sound. Afterward the certificate of the veterinarian was given to that effect. The State board of health and its veterinarian were refused entrance to the stable. Owing to alleged or possible defects in the law a new one was passed. It was not, therefore, until April that the board was again able to secure undisputed entry. On the first visit this horse was found with symptoms of glanders, and a *post-mortem* examination fully attested the former diagnosis. There were other suspicious cases, and it was found necessary soon after to condemn two more. The disease is now in such a chronic, and in some cases probably in such a concealed form, that it is claimed by some that nothing short of a destruction of all the live-stock and the buildings will eradicate it. Occasional cases are now occurring in other parts of the city. The whole history of these cases illustrates the persistent infection of the malady, and shows how delay by the interposition of legal obstacles may result in entailing upon an entire city the continuance of a disease which could have been eradicated in the start.

As there is much difference of opinion as to the possibility of the spontaneous development of glanders in stables where horses are overworked or illy kept, it seems very desirable that the General Government should institute a series of experiments and investigations for the purpose of determining this point. The disease is of a very threatening character to the great industries which so much depend on this class of animals. As, too, it is admitted that stables which have contained affected horses, and all the harness used come to be fomites or foci of communication, there is need of the most precise directions as to the choice of disinfectants and their accurate and successful use. It is doubtful whether this can be left to owners or even to the general veterinarian. It is better rather to commit it to those who understand all the details of fumigation, inhalation, and the washing and saturation of all exposed surroundings and material.

Our experiences in this State for the past year with pleuro-pneumonia, glanders, and hog cholera are sufficient to emphasize the immense importance of close investigation of all contagious animal diseases by the General Government. There is need not merely of hurried inquiry into alleged outbreaks of contagions, and the rapid application of methods of isolation, destruction, or quarantine, but of scientific and laboratory investigations and such skilled observations and experiences as will prevent their occurrence. The notable results that have already occurred from the combined inquiry and insight of medical and veterinary experts into the general development and life-history of these marauding pestilences, is enough to assure us that our neglect will be culpable, if we fail to protect our great commercial and industrial interests by saving as far as possible the live-stock of our country from invasions as disastrous as those of pestilence and famine.

SOUTHERN CATTLE FEVER AT THE CHICAGO UNION STOCK-YARDS.

Hon. GEORGE B. LORING,
Commissioner of Agriculture:

SIR: On the 29th day of July last, on my way to Chicago, I read an account in the Daily Tribune of that city of an outbreak of disease among cattle at the stock-yards. I concluded, therefore, to proceed to the yards and ascertain if possible the nature of the disease.

After my arrival in the city I made inquiries, but had considerable difficulty in gaining any positive information concerning the location of the diseased stock. However, I finally found the affected animals in block 22, at the extreme southern limits of the pens used for hogs. The inclosure was a small field, and contained about 500 head of cattle, ranging in age from one and a half to four years. The majority of them were half-breed Texans. Many were near the point of death, and, by order of Mr. William Mitchell, agent for the Illinois Humane Society, were shot. Among others, the various stages of an acute disease were manifested.

I formed the acquaintance of Mr. Mitchell and of Mr. Leek, the latter assistant inspector to Mr. Lamb. From these gentlemen I obtained the following history:

On the previous day seventeen car-loads of cattle arrived at the yards, consigned by Frazer & Oberly, of Kansas City, Mo., to Keenan & Hancock, of Chicago. These cattle were supposed to have come from Fort Reno, via. Kansas City. Out of the 530 head, 64 were found dead on their arrival at the yards, and a large number were in such a condition that they were ordered to be shot; the remainder were driven into the yard where I found them.

On the 29th another shipment was received from Barnard, Mo., consigned to Fisher, Evans & Co. This lot numbered 91 head, 14 of which were found dead on their arrival, and many others were diseased. These were placed with the preceding lot of cattle, and were of the same class and grade of stock. During the day many new cases of sickness appeared, and quite a large number died or were shot. I learned that Dr. De Wolfe, health commissioner of the city of Chicago, and Dr. N. H. Paaren, State Veterinarian of Illinois, had been at the yards in the morning making examinations and autopsies, but unfortunately they had left before my arrival I made a close inspection of the diseased cattle and arrived at the conclusion that they were suffering with the so-called Texas or Southern cattle fever. Before I left the yards I was informed that "twenty-three more car-loads were on the way from Kansas City to Chicago having sick cattle on board." I returned to the city, but too late to see either Drs. De Wolfe or Paaren. Next morning (July 30) I went to Dr. De Wolfe's office, but he had not yet arrived; I also visited the residence of Dr. Paaren, where I was informed that he was in the city, but could not learn where to find him.

I visited the stock-yards a second time, and called upon Superintendent Williams. He informed me that all of the cattle in the diseased lot, which were able to travel, had been driven to O'Malley's slaughter-house, and were there to be killed under the supervision of Dr. De Wolfe and his agents. Mr. Curzon accompanied me to the above-named slaughter-house. On our arrival we found a majority of the diseased animals slaughtered, and the work rapidly progressing. Inspectionby the agents of Dr. De Wolfe, under the direction of Inspector Lamb, were made of each animal as the internal organs were removed from thecarcass. Mr. Lamb kindly gave me permission to examine as many of the cattle as I desired, and called my attention to, and showed me in, what manner they conducted the inspection and disposed ofthe carcasses—both the healthy and infected. Forty per cent. of the animals slaughtered in my presence exhibited the characteristic lesions of the Southern cattle fever, and were accordingly condemned and ordered to the rendering tanks. All animals which presented any of the following conditions were condemned as being unfit for consumption, viz: Enlarged spleen, engorged and enlarged kidneys, bloody urine in the bladder, and yellow discoloration of the fat.

In my estimation the meat inspection was efficiently and conscientiously performed.

Finding nothing further to be done by me, as the State and city authorities had taken the matter in hand, I took my leave. Returning to Chicago on the 5th of August I called upon Dr. Paaren, who supplied me with a copy of his report which he had forwarded to Governor Hamilton. He could give me no other information then than that the cattle had been originally shipped from Fort Reno. Subsequently I learned that the whole of the two lots and the 23 car-loads which were reported on the way on the 29th of July had been grazed all summer along the northern line of the Indian Territory on a range claimed by Frazier & Oberly, and that they were driven to Caldwell, Kans., placed on board the cars, and taken to Kansas City, Mo. On the day of their arrival at Kansas City several of them died; on the following day more died. The stock men became alarmed and rushed them off to Chicago. The 23 car-loads were not allowed to enter the Union Stock-Yards on their arrival (July 31), but were immediately taken to the slaughter-house, where they met the same fate as had the previous shipments. I was informed by Dr. De Wolfe on the 6th of August that out of 964 head received at Chicago—dead and alive—720 were condemned and delivered to the rendering tanks.

Respectfully submitted.

M. R. TRUMBOWER,
Veterinary Inspector.

STERLING, ILL., *August* 15, 1884.

OUTBREAK OF SWINE PLAGUE IN MARYLAND AND VIRGINIA.

Hon. E. A. CARMAN,
Acting Commissioner of Agriculture:

SIR: In compliance with your request I left this city on the morning of the 21st instant for the purpose of investigating and determining the extent to which swine plague exists among hogs in the counties bordering on the Upper Potomac River. I did not find any dead hogs in the river near Harper's Ferry, nor could I hear of any suffering with the disease in this vicinity on the morning of my arrival. At this point it was a matter of doubt with me whether I should go up the Shenandoah River, which joins the Potomac here, or continue up the last-named stream. However, after diligent inquiry I thought it best to follow up the Potomac. From Harper's Ferry I traveled a northerly course on foot for 3 miles to Engle's Switch, on the Baltimore and Ohio Railroad. Here I learned that many hogs had died of the disease during the months of June, July, and August.

Throughout Jefferson County, West Virginia, a large number of animals had succumbed to the disease, some farmers having lost almost their entire herds. During the three days of my investigations at about all the points I visited I found that the disease had abated, but in many cases this was owing to the lack of victims. After my first day's experience I found it impossible to keep anything like an accurate record of the losses, but it is safe to say that thousands of hogs of all ages, but more especially shoats or young animals, have perished during the prevalence of this epidemic in the counties bordering the Potomac River.

From Engle's Switch I continued my journey on foot through Jefferson to Duffield, in the same county, and from there on to Shenandoah Junction. I had no trouble in getting information as to the extent and destructiveness of the epidemic. Everybody seemed to have suffered to a greater or less extent, and all were anxious to furnish whatever information they possessed.

On the 22d instant I made an inspection of the Potomac River at Shepherdstown, Jefferson County, West Virginia, a locality about 12 miles above Harper's Ferry. In this locality the farmers have lost heavily, some of them as high as 100 head. Mr. Wrench, the keeper of the bridge which crosses the river here, informed me that many of the people dumped their hogs into the Chesapeake and Ohio Canal until the canal men made complaint of the terrible stench. After this they threw them into the river. I crossed the bridge and walked along the canal on the Maryland side for about a mile in the direction of Harper's Ferry. Here, at a dam near the Cement Works, I found a number of dead hogs. It has been the practice here to remove the dead hogs—after they had drifted down in large numbers—and allow them to pass below. Persons here admitted that they had thrown their dead hogs into the canal, and stated that they had seen dead mules in the same stream at all seasons of the year.

I am informed by reliable parties that many dead hogs have been cast into the stream between Shepherdstown and Antietam Creek, a distance of some 3 miles. No one seems to have objected or thought the matter worthy of investigation. It is true but very few dead hogs are to be found at the present time polluting the streams, but this is due more for a lack of hogs for the plague to work upon than the disposition of the people to burn or properly bury the carcasses.

From Shepherdstown, I walked to Sharpsburg, Md., where I learned that the disease, though it had prevailed in that vicinity, had not been so destructive as elsewhere. A Mr. Reefe, who lives 2 miles north of this place, had lost twenty head. I here learned that the disease was prevailing quite extensively in the vicinity of Charlestown, Jefferson County, West Virginia, but on visiting that locality I found it had very much abated. I was told by the people of this county that their losses had been very heavy; that the disease had been very destructive to the farmers along the line of the railroad to Harper's Ferry on the one side and to Winchester on the other.

On the 23d instant I visited Point of Rocks, Frederick County, Maryland. The greatest number of hogs seem to have died in this vicinity during August and early part of this month. Hundreds are reported to have died in the county. The disease is said to have been very destructive in the vicinity of Frederick City.

In Loudoun County, Virginia, the disease had been as destructive as elsewhere along the borders of the Potomac. Many of the animals that died in this county were thrown into the river opposite Point of Rocks. But the large majority of the carcasses that have been seen in the river here were thrown into the canal and river in the vicinity of Shepherdstown and Antietam Creek, and were drifted down by the current. Since the visit of Dr. Townshend, who posted bills between Washington and Harper's Ferry, no dead carcasses have been thrown into the canal or river between those points.

At Germantown, Montgomery County, Maryland, and vicinity, I learned that the disease had been very destructive, but had about spent its force for the lack of material to work upon. All the farmers west of the Baltimore and Ohio Railroad had lost almost all their hogs. The disease made its appearance in this locality in June last. Some farmers lost as high as eighty head, and before the disease abated it was estimated that at least two-thirds of all the animals in the vicinity had died.

I did not visit Rockville and Sandy Springs, but I learned that the plague had made its appearance in the vicinity of those places.

To conclude: The results of my investigation lead me to believe that thousands of hogs have died within the past two or three months of swine plague in the territory embraced within the counties of Montgomery, Frederick, and Washington, in Maryland, and in Jefferson and Loudoun, in Virginia.

Respectfully submitted.

W. H. ROSE,
Veterinary Inspector.

WASHINGTON, D. C., *September* 26, 1884.

OUTBREAK OF CONTAGIOUS PLEURO-PNEUMONIA IN DELAWARE.

On the 7th of January instant the Department was informed by Mr. George G. Lobdell, of the Lobdell Car-Wheel Company of Wilmington, Del., of a serious outbreak of contagious pleuro-pneumonia in a herd of cattle belonging to that company. His letter also indicated that the disease was prevailing in other localities in the State, and was of such a menacing character as to demand the immediate attention of the State authorities, and for the purpose of securing such assistance as might be necessary in preventing the further spread of the plague, he proposed to visit and consult Governor Stockley on the following day.

Two animals belonging to the above-named company had died and three others were sick.

On the 10th instant Mr. J. Thomas Budd, statistical agent of the Department for the State of Delaware, wrote that contagious pleuro-pneumonia was prevailing to an alarming extent in the northern portion of New Castle County, and inclosed the following article from Every Evening, a daily paper published at Wilmington:

DREADED CATTLE PLAGUE—PLEURO-PNEUMONIA SPREADING FAST IN THE COUNTY.

Almost every day since Every Evening first warned the public of the prevalence of pleuro-pneumonia among the cattle of New Castle County have new cases of the infection been reported. At first it was thought that the disease was confined to two or three farms in Brandywine Hundred, but it is now known to exist in various other parts of the county, and to such an extent as to awaken the greatest alarm among owners of cattle. The prevalence of the dreaded plague in George G. Lobdell's dairy, near the city line, is reported at Every Evening office this morning. Ward B. Rowland, veterinary surgeon, has examined the herd, and announces that eleven of the cows are suffering with the pleuro-pneumonia. Two or three of the affected animals are suffering with the malady in an acute form, and in the others the disease is rapidly developing itself. Some cases have already in this dairy proven fatal, and the cows were hauled through the city and out the Newport pike to a marshy meadow, where they were deposited, to be eaten by carnivorous scavengers.

It is, however, but just to the owner of the cattle to state that he was in ignorance of the exceeding malignant nature of the ailment. In taking the carcasses of the dead animals along the road the malignant germs of the disease have perhaps been sown and will probably develop in time in other cattle. The milk of the dairy, which is usually sold to customers in this city, is not sold here any more. Every precaution is being taken to prevent the further spread of the disease from this source, and the herd are under quarantine and the affected members isolated a safe distance from the others. The governor has been formally notified of the infection of this herd, and has intimated that he will give the matter his immediate personal attention.

Governor Stockley, it is fully anticipated, will be in Dover on Monday, and urge upon the legislature the necessity of taking prompt measures to stamp out the disease. He will probably get the sentiment of the members on the subject, and anticipate their final action by issuing a sort of order covering the necessities of the cases now in hand. The legislature will then enact laws to meet all emergencies with relation to stamping out the malady.

A communication from William Dean to-day says:

"I see by Every Evening that the much-dreaded cattle disease, pleuro-pneumonia, is within the borders of our State. Your citizen, Dr. L. P. Bush, has written to me, as chairman of the State executive committee of Patrons of Husbandry, in reference to the matter, but our organization is entirely powerless to take any effective measure to stamp out the much-dreaded disease. The only remedy we have to eradicate it must come through the authority of our legislature, which now, fortunately, is in session. Knowing a majority of the members as I do, I have entire confidence in their wisdom and determination to act for the best interests of the State, and believe they will take immediate action on the matter. As an evidence of their disposition to care for our wants, the Hon. S. F. Armstrong came to my house to-day to see me in regard to the matter, I being confined to my house with sickness. He no doubt will have the matter in good shape to present to his fellow-representatives on the assembling of the legislature next Monday, when I hope no time will be lost in adopting such measures as in their wisdom will at once put a stop to a disease that threatens not only the destruction of a large amount of property, but also the health of our entire people."

The necessity of a State physician, to whom all matters may be referred for his action or concurrence with other officials, is most felt. Dr. Ward B. Rowland, of this city, who is a graduate of the New York College of Veterinary Surgery, has thus far been consulted by the owners of the infected herds. The fact of his being the only veterinary surgeon in Delaware who has graduated from a regular college, and who has had considerable experience in pleuro-pneumonia cases, makes his advice in the matter much sought after just now.

Mr. Lobdell, in his communication of the 7th instant, having asked if this subject came under the jurisdiction of this Department, was promptly informed by the Commissioner that he would at once direct two Veterinary Inspectors to make an investigation as to the extent to which the disease prevailed among the cattle of Delaware, and would advise the governor of his action and ask his co-operation in such "disinfection and quarantine measures as may be necessary to prevent the spread of the disease from one State to another," as authorized by the act of Congress approved May 29, 1884, providing for the establishment of the Bureau of Animal Industry. The following is a copy of the letter addressed to Governor Charles C. Stockley:

DEPARTMENT OF AGRICULTURE,
Washington, D. C., January 10, 1885.

SIR: It is provided in section 3 of the act of Congress approved May 29, 1884, of which I inclose a copy, that "whenever the plans and methods of the Commissioner of Agriculture shall be accepted by any State or Territory in which pleuro-pneumonia or other contagious, infectious, or communicable disease is declared to exist, or such State or Territory shall have adopted plans and methods for the suppression and extirpation of said diseases, and such plans and methods shall be accepted by the Commissioner of Agriculture, and whenever the governor of a State or other properly constituted authorities signify their readiness to co-operate for the extinction of any contagious, infectious, or communicable disease in conformity with the provisions of this act, the Commissioner of Agriculture is hereby authorized to expend so much of the money appropriated by this act as may be necessary in such investigations, and in such disinfection and quarantine measures as may be necessary to prevent the spread of the disease from one State or Territory into another."

The existence of pleuro-pneumonia in Delaware, and the recent shipments of cattle from infected herds in other States, has brought up the question of quarantine, and as the co-operation of State authorities is necessary before expenditures for quarantine purposes are allowed by this law, I desire information as to whether it has adopted any "plans and methods for the suppression of" this disease, or whether, as governor, you are willing or authorized by law to "co-operate" within the meaning of this section.

As the question is a very urgent one, an early reply is solicited.

Very respectfully,

GEO. B. LORING,
Commissioner of Agriculture.

Governor CHARLES C. STOCKLEY,
Dover, Delaware.

To this letter Governor Stockley promptly responded as follows:

STATE OF DELAWARE, EXECUTIVE DEPARTMENT,
Dover, Del., January 16, 1885.

SIR: Your favor of the 10th instant, with copy of the act establishing a Bureau of Animal Industry, was duly received. In reply I would most respectfully say that, in compliance with the laws of this State, I have issued a proclamation, and in compliance with the same have appointed Ward B. Rowland, of Wilmington, Del., State veterinary surgeon. I can see nothing in either act which would prevent us from co-operating, and I shall be very much pleased to do so.

Inclosed find copy of our law. If, upon examination, you think any further legislation on our part is necessary to enable us to co-operate fully in the matter, I shall be pleased to hear from you.

I remain, very respectfully, yours,

CHARLES C. STOCKLEY.

Hon. GEORGE B. LORING,
Commissioner of Agriculture.

The following is a copy of the proclamation issued by Governor Stockley, which is accompanied by a copy of the law of the State of Delaware for the suppression of contagious and infectious diseases of domestic animals:

PROCLAMATION.

EXECUTIVE DEPARTMENT,
State of Delaware:

Whereas, reliable information has been received that the disease known as pleuro-pneumonia exists among cattle in this State. Now, therefore, I, Charles C. Stockley, governor of the State of Delaware, by virtue of the act of the general assembly entitled "An act to prevent the spread of contagious or infectious pleuro-pneumonia among the cattle of this State," passed March 29, 1881, declare and proclaim that said infectious or contagious disease exists in this State, and do order and direct all persons to seclude all animals in their possession that are affected with such disease or have been exposed to the infection or contagion thereof, as the nature thereof may, in their judgment, render necessary or expedient. It is further ordered and directed that any premises, farm or farms, where such disease exists or has existed, shall be put in quarantine, so that no domestic animal be removed from such places so quarantined. Such regulations will be prescribed and such officers appointed as may be necessary for the proper execution of the law. All sheriffs and deputy sheriffs are hereby ordered and directed to carry out and enforce the orders of this proclamation and to see that all of the provisions of the aforesaid act are fully obeyed.

Any person disobeying the orders of the governor, sheriffs, or deputy sheriffs, made in conformity to the above-mentioned act, are liable to a fine not exceeding $500, or to imprisonment not exceeding one year.

In witness whereof the great seal of the State of Delaware is affixed.

[L. S.]

Witness, Charles C. Stockley, esq., governor of the said State at Dover, the thirteenth day of January, in the year of our Lord, one thousand eight hundred and eighty-five, and in the year of Independence of the United States the one hundred and ninth.

CHARLES C. STOCKLEY.

By the governor:
WILLIAM F. CAUSEY,
Secretary of State.

Act of March 29, 1881.

SEC. 1. That whenever the governor of this State shall receive reliable information that the disease known as contagious or infectious pleuro-pneumonia exists among cattle in this State, he shall have power, and is hereby authorized, to issue his proclamation, stating that the said infectious or contagious disease exists in this State, and warning all persons to seclude all animals in their possession that are affected with such disease, or have been exposed to the infection or contagion thereof, and ordering all persons to take such precautions against the spreading of such disease as the nature thereof may, in his judgment, render necessary or expedient; to order that any premises, farm or farms, where such disease exists or has existed, to be put in quarantine, so that no domestic animal be removed from such places so quarantined, and to prescribe such regulations as he may judge necessary or expedient to prevent infection or contagion being communicated in any way from the places so quarantined; to call upon all sheriffs or deputy sheriffs in this State, to carry out and enforce the provisions of such proclamations, orders, and regulations; and it shall be the duty of all the sheriffs and deputy sheriffs to obey and observe all orders and instructions which they may receive from the governor in the premises; to employ such and so many medical and veterinary practitioners, and such other persons as he may, from time to time, deem necessary, to assist him in performing his duties as set forth in this act, and to fix their compensation, when in his judgment it shall be deemed necessary to order all or any animals coming into this State to be detained at any place or places for the purpose of inspection and examination; to prescribe regulations for the destruction of animals affected with the said infectious or contagious disease, and for the proper disposition of their hides and carcasses, and of all the objects which might convey infection or contagion (provided that no animal shall be destroyed unless first examined by a medical or veterinary practitioner in the employ of the governor, as aforesa'd); to prescribe regulations for the disinfection of all premises, buildings, and railway cars, and of objects from or by which infection or contagion may take place or be conveyed; to alter and modify, from time to time, as he may deem expedient, the terms of all such proclamations, orders, and regulations, and to cancel or withdraw the same at any time.

SEC. 2. That in any case where, in the opinion of the governor, it may be deemed necessary to destroy any diseased animal or animals, the said governor shall select and appoint three judicious and impartial citizens of this State to view, and after such view, to assess any such diseased cattle at what would be their real value in money were they not so affected; and in case said cattle shall be destroyed, as provided in the first section of this act, then upon certificate of such assessment, duly signed and attested by said assessors or a majority of them, being presented by the owner or owners of such destroyed animals to the State treasurer of this State, the said State treasurer is hereby authorized to pay to such owner or owners the sum of money equal to two-thirds of the amount of the aggregate assessment upon said cattle so destroyed.

SEC. 3. If any person shall sell or dispose of any animal or animals, known to be affected with pleuro-pneumonia, or known to have been exposed thereto within one year prior to such sale or disposal, without due notice to such purchaser that said disease exists in said animals, or that they have been exposed thereto, as aforesaid, he shall be deemed guilty of a misdemeanor, and shall be punished by fine not exceeding $500, or may, in the discretion of the court, be imprisoned for a term not exceeding one year.

SEC. 4. If any one knows, or has reason to suspect, that said disease exists among the cattle in his possession or under his care, he shall forthwith give notice thereof to the governor, and for failure so to do, shall be punished by fine not exceeding $500, or by imprisonment not exceeding one year.

SEC. 5. Any person disobeying the orders of the governor, sheriff, or deputy sheriff, made in conformity to this act, or any person driving or transporting any neat cattle contrary to the regulations made and published as aforesaid, shall be punished by fine not exceeding $500 or by imprisonment not exceeding one year.

SEC. 6. That all the necessary expenses incurred under the direction or by the authority of the governor in carrying out the provisions of this act, shall be paid by the State treasurer upon proper certificate of the assessors of diseased cattle which have been destroyed or upon warrant duly made and signed by the governor, on the State treasurer, for all expenses incurred under this act other than the payment for cattle destroyed.

SEC. 7. That animals coming from a neighboring State that have passed a veterinary examination in said State, or have been quarantined and discharged, the owner or driver being provided with a genuine certificate that such animals are not infected with pleuro-pneumonia, shall not be subject to the provisions of this act.

SEC. 8. That all the provisions of this act applicable to the disease known as pleuro-pneumonia be also, and are hereby, extended to and made applicable to all other infectious or contagious diseases which are virulent and fatal in their nature.

Drs. W. B. E. Miller and C. K. Dyer, Veterinary Inspectors of this Bureau, commenced an inspection of the herds of this State on Monday, January 19, 1885.

TEXAS FEVER—TUBERCULOSIS—GLANDERS.

Hon. **George B. Loring**,
Commissioner of Agriculture:

Sir: On the morning of the 3d I drove to Polo, Ogle County, a distance of 18 miles. Soon after my arrival your message of the same date was transmitted to me by telephone from Sterling. I called upon Barber Bros., who informed me that a lot of cattle had been brought from Chicago in the latter part of October, and were pastured for a few days next to one of their farms; that on the morning of the 27th of October they found one six-months-old calf dead in their field and had it buried, but on the following day, after hearing that the foreign cattle were dying with pleuro pneumonia, they became alarmed, exhumed the dead calf, and had Dr. J. H. Judson make a *post-mortem* examination. Dr. Judson, after the examination, was unable to state the cause of death. On the following Saturday morning, November 1, Mr. George Brand, who had furnished pasturage for the Chicago cattle in a field adjoining one wherein his cattle were grazing, also found a yearling heifer dead in the field. Mr. Brand covered it with hay, and went to see Barber Bros. On the 2d they sent a telephone message requesting me to make an examination of the latter animal; at the same time they telegraphed to you for an investigation.

Barber Bros. stated that their calf had been weakly and suffered with a chronic diarrhea all summer, and that they did not expect it to survive cold weather. On the 26th a cold drizzling rain set in, and they supposed congestive chills caused the death of their animal.

Bryant H. Barber took me to Mr. Brand's farm, and there I examined his dead heifer. With the exception of the digestive organs I discovered no abnormal conditions, either of blood, urine, or other fluids of the body. The living membranes of all four stomachs were intensely congested, and deep and large ulcerations existed in the mucous membrane of the fourth. In the absence of congested liver or spleen, discoloration of fat, bloody urine, &c., I did not believe it died with Texas fever, but that some violent irritant poison caused its death. This animal had not been on any of the ground on which, or over which, the Chicago cattle had grazed. Mr. Brand, on the morning of the 1st, after finding the dead one, carefully watched all of his other cattle (30 head), gave them salt to eat, and noticed that many of them coughed. Mr. Barber also was watching their spring calves (60 head), and discovered that many of them had a nasal discharge, but no cough. At the time of my visit I heard no cough, nor observed any nasal discharge. On our return to Polo I met Col. M. D. Swift, attorney-at-law, and Henry Metz, and obtained from them the following history:

On the 15th of October, Solomon Metz, a dealer in cattle, purchased at the Union Stock-Yards, Chicago, 81 head of yearling and two-year-old cattle which had been brought from Detroit, Mich. On the 16th Metz shipped them at Chicago, and arrived with them at Maryland Station, 6 miles north of Polo, on the morning of the 17th; he then drove them to Mr. Brand's, 1½ mile north of Polo. On the 2d he removed them, together with about 30 head of natives, from Mr. Brand's field, and drove them west 8 miles, and placed them in a field belonging to Mr. George Shaver. While they remained here he sold 20 head of them to Mr. Morgan, of Elkhorn Grove. On the 25th he drove the remainder north 10 miles to the farm of Mr. Jacob Micheals, in Carroll County; there he sold, on the same day, 23 head to Mr. Heimbach, residing 8 miles south of Lanark. On the following Monday, the 27th, he sold 16 head to Mr. Micheals, and then drove the rest of them 5 miles north to Shannon, and sold 21 of them to Elias Good, who resides 2 miles north from Shannon. On their way to Shannon from the Micheals farm four of the animals became so weak that they were left lying along the road, and all four of them were dead next morning. The remainder of the cattle were reshipped to Chicago on the 28th.

On the 5th of November I drove to the residence of Mr. Elias Good, and there learned that one of the cattle purchased from Metz died on the 28th and a second one on the 30th of October; a third one, which had been sick, had recovered. He said: "They became weak, frothed at the mouth, voided bloody urine; manure was hard, and they died without a struggle." From Good's I drove to Micheals'. He lost one on the 28th, one on the 29th, and another one on the 30th of October. A fourth was found sick at noon on the day of my visit. At 4 o'clock p. m. it (a two-year-old steer) had a temperature of 104⅖° Fahrenheit, pulse weak, limbs cold, nose dry, and persisted in lying down; when he got on his feet he walked off very stiff and weak. Mr. Micheals stated that the two of the three which died passed pieces covered with blood-clots and mucus, frothed at the mouth, and ate some grass almost up to the time of death. On the 30th of October Dr. Judson made a *post-mortem* examination of the one that died on the 29th, but failed to discover the cause of death and the nature of the disease. Micheals, however, stated that the bile was very thick and black, and the fat was of a rich yellow color.

After taking into consideration the statements of Good and Micheals, I had no hesitation in diagnosing the disease to have been the Texas fever. I then drove toward Mr. Heimbach's place, and met a man on the road who is a close neighbor to the former. He told me that Heimbach had lost two out of the number purchased from Metz. I then turned back and went over the road on which the cattle had been driven by Metz; also followed their route back to Maryland Station, but failed to discover any evidence that they had scattered the disease. Hence I have reason to believe that no Southern cattle were among the lot of 81 purchased at Chicago, and that no cause for alarm exists as far as communicating the disease to other native cattle may be concerned.

I arrived home on Friday evening, the 7th instant, when I received your telegram of the 6th, requesting me to go to Waterloo, Iowa, and there to examine a heifer belonging to Mr. Edward Camp. I left for Waterloo on the 8th. Mr. Camp purchased from Richardson Bros., of Davenport, Iowa, two yearling Jersey heifers on or about the 20th day of July. He took them home and placed them in a field with 20 head of other cattle, twelve of which were Jerseys. Brisk, one of the two purchased, did not look well when he brought her home. About six weeks afterwards he discovered that she did not keep with the herd, and was lying down most of the time; when made to move she had severe fits of coughing, as if she was choking, breathed very hard and quick. He removed her from the field to his barn, and has kept her away from the other cattle ever since. At that time she did not eat anything for several days. After she commenced to eat again her cough gradually became less frequent and harassing. I found her looking fairly well, temperature 100½° F., breathing nearly normal; a crepitant rale was audible over the whole of both sides of the chest; the left side was normally resonant, while the lower part of the right lung emitted dull sounds in several small circumscribed places on percussion; pressure on the trachea at the point of entrance into the chest caused pain and coughing. The cough was moist and full. The crepitation resembled emphysema in horses, and I attributed it to the same condition in this heifer. She evidently had a severe attack of bronchitis, and possessing a weak constitution, is now developing consumption, which she probably inherits. None of the cattle she has been grazing with during the space of six weeks manifest any cough or other evidence of disease.

Mr. Dwight Ryder, a resident of Waterloo, volunteered to take me out of town to see a horse belonging to Mr. W. W. Straher, which they supposed was affected with glanders. I accepted the invitation and examined the 8-years old bay horse of Straher's. He manifested characteristic enlargement of the submaxillary lymphatics, ulceration on the schneiderian membrane in both nostrils, a yellow sticky discharge, more profuse from the right than the left side. I pronounced it a case of chronic glanders, and advised the owner to kill and bury him without any unnecessary delay. Mr. Straher traded for this horse about two months ago, and he was then running at the nose. Mr. Joseph Heileman, of Waterloo, from whom he obtained this horse, killed a mare affected with glanders and farcy last March. Professor Stalker authorized the killing of two horses which were glandered several months ago. These horses were owned about 2 miles north of Waterloo, and were driven to the town very frequently while so affected.

Yours, obediently,

M. R. TRUMBOWER,
Vetinary Inspector.

STERLING, ILL., *November* 10, 1884.

CANADIAN RESTRICTIONS ON AMERICAN CATTLE.

The Canadian Government, in accordance with a decree of its council, adopted on the 8th day of September last, has issued the following rules and regulations relative to the importation into, and the transportation through, that Dominion, of American cattle:

Whereas the disease of pleuro-pneumonia prevails among neat cattle in the Western State of Illinois as well as in other more eastern of the United States, and there is reason to believe that neat cattle for breeding purposes have been sent from the State of Illinois to more western States and Territories;

On the recommendation of the minister of agriculture, and under the provisions of the act of the Parliament of Canada, 42 Victoria, chapter 23, entitled "An act to provide against infectious or contagious diseases affecting animals," made applicable to the Northwest Territories by proclamation in 1883;

His excellency, by and with the advice of the Queen's Privy Council for Canada, has been pleased to order and it is hereby ordered, that the importation of neat cattle now permitted from the United States and Territories into the Province of Manitoba and the Northwest Territory of Canada, be, and the same is hereby prohibited, except on the following conditions, namely:

1. At Emerson, in Manitoba, or the points of Fort Walsh and Fort McLeod, in the provisional districts of Alberta and Assiniboia, or such other point or points as may be hereafter indicated by the minister of agriculture.

2. For stock or breeding purposes, neat cattle which have been brought to the Canadian frontier for importation, may be allowed to cross, subject to the regulations hereinafter recited.

3. For transit, from west to east, through the provisional districts of Alberta and Assiniboia, and the Province of Manitoba, via. Emerson or Gretna, to the State of Minnesota, neat cattle may be allowed to cross the Canadian frontier at the points of Fort Walsh and Fort McLeod aforesaid, subject to the regulations hereinafter recited.

4. At Emerson, such cattle coming from the East shall not be allowed to cross the Canadian frontier unless after inspection by a duly authorized veterinary surgeon, appointed by the Minister of Agriculture, they shall be declared free from contagious disease, and also from well-founded suspicion thereof; and further, such cattle shall be subject to a quarantine of sixty days, or such other period as may appear to the Minister of Agriculture advisable.

5. Any cattle desired to be entered at the points of Fort Walsh and Fort McLeod aforesaid, whether for stock or breeding purposes or for transit, shall be inspected by a duly authorized veterinary surgeon appointed by the Minister of Agriculture, and shall not be allowed to cross the Canadian frontier unless they are declared by such surgeon to be free from contagious disease, and also from well-founded suspicion thereof.

6. The owner or owners of any such cattle desired to be entered at any of the points aforesaid, shall, on making application for entry, produce a duly attested certificate, indicating the State or Territory and particular locality from which they may have been brought.

7. The importer of such cattle shall pay a fee, graded on a scale hereto annexed, to the customs officer, or other person duly authorized to act as such, for defraying the expense of such inspection, the cattle not being allowed to cross the Canadian frontier until such fee is paid; that is to say, for one animal, $1; 5 animals and under, 50 cents each, but total fee for over 5 animals not less than $2.50; 10 animals and under, 30 cents each, but total fee for over 10 animals not less than $3; 20 animals and under, 20 cents each, but total fee for over 20 animals not less than $4; 50 animals and under, 12 cents each, but total fee for over 50 animals not less than $6; over 50 animals, 10 cents each.

8. No car which has been loaded with cattle in the United States and crossing the Canadian frontier shall be allowed afterwards to carry Canadian cattle.

9. No car nor trains carrying such United States cattle in transit from west to east between the points above named shall be allowed to be or remain shunted in close proximity to any Canadian cattle.

10. Every car containing such cattle in transit between the points above mentioned shall be kept as far as possible apart from cars or trains containing Canadian cattle or Canadian goods.

11. No car containing such United States cattle in transit between the points above named shall form any part of a train carrying Canadian cattle.

12. Every car or train carrying cattle in transit from west to east between the points hereinbefore named shall stop at such fixed place or places as shall be named by the Minister of Agriculture for the purpose of rest, feeding, and watering, and such place or places shall be declared "infected" within the terms of "*The animal contagious diseases act*, 1879," being strictly isolated, and all communication with them prohibited, except by the officers and men in charge of the trains or in charge of such infected place or places.

13. Every car which has been used for carrying animals from the United States or Territories in transit through the Districts of Alberta, Assiniboia, or the Province of Manitoba via. Emerson or Gretna, shall be thoroughly cleansed and disinfected before re-entering the Province of Manitoba, in such manner as shall be ordered by the Minister of Agriculture.

JOHN J. McGEE,
Clerk, Privy Council.

A telegram having announced the suspension of this order, the Department at once addressed Hon. Mr. Pope, Minister of Agriculture for the Dominion of Canada, for more definite information on the subject. The following, which was promptly received, is an extract from his reply to the inquiry of this Department:

I have to inform you, in reply to your letter of the 15th inst. (December, 1884), that the regulations authorized by order in council of the 8th of September last, are in force as respects cattle from Montana entering at the points of Forts Walsh or MacLeod for transit, from west to east, through the provisional districts of Alberta and Assiniboia, and the Province of Manitoba via Emerson or Gretna, to the State of Minnesota.

As respects cattle entering the Province of Manitoba at the point of Emerson, the quarantine will be suspended during the winter months, but the inspection enforced in the case of cattle which arrive for entry and those found healthy will be allowed to pass on.

OUTBREAK OF GLANDERS AT MIAMI, MO.

Hon. Geo. B. Loring,
Commissioner of Agriculture:

SIR: In obedience to your telegram of October 21, I left home on the 22d, and arrived at Miami, Mo., on the evening of the 23d. I inquired for Mr. G. R. McDaniel, and was informed that he was absent from home, attending a Baptist convention at Marshall, Mo. I sent a telephone message informing him of my arrival, and the next morning Mr. R. S. Ireland, secretary of the Miami Anti-Horse Thief Association, presented himself, having been requested by Mr. McDaniel to attend me in the investigation of a disease among horses, supposed to be glanders. Mr. Ireland procured a team and took me to Mr. Alfred Wheeler's farm, distant about two miles from town. Mr. Wheeler owns a five-year-old brown mare, which has been suffering with influenza since early in the summer, and has continued to discharge at the nose since that time. I made a careful examination, but could not discover any indication of glanders. I examined five other horses and mules on the place, but found them all healthy and well. This mare of Wheeler's was one of the suspected cases. We returned to town, and after dinner, accompanied by Mr. Willis Hayes, a livery-man, I was taken six miles southeast to the farm of Mr. Robert Smith, where some more of the suspected cases were located. Mr. Smith, who had been very uneasy about his horses, was highly gratified when I made my business known to him. He took me out to a field on his farm, and there showed me two colts, one two and the other three years old. Examination revealed subacute glanders. Large and numerous glanders ulcers were visible in both nostrils; difficult and snuffling breathing; dullness was elicited on percussion over the sinuses, and in one case the ulcerations had perforated the *septum nasi*, and the discharge was profuse, fetid, and mixed with blood. Extensive induration of the submaxillary lymphatics existed in both cases. These colts were supposed to have had the distemper in the spring, and had not recovered therefrom, but gradually grew worse, and finally became greatly debilitated. Mr. Smith, very fortunately, had turned them into a field by themselves early in the spring, and thereby avoided contamination of his other horses and mules. However, two young mules, belonging Mr. C. E. Sheppard, had been with these colts at several different times during the spring and summer months. They became affected with glanders. When Mr. Sheppard became suspicious of the disease he turned them into Smith's field with the colts, believing that they had there contracted the disease. These mules were in the said field with the colts when I arrived, and I also examined them. I found them in even a worse condition than the others. The older one (four years) had extensive swellings about the nostrils and nasal bones, in addition to very extensive destruction of the pituitary membranes and deeper structures, and one of them was reduced to a mere skeleton. As these mules had been on the owner's farm, and there came in contact with a number of his horses and mules, I deemed it my duty to examine all of his stock. We drove two miles to Mr. Sheppard's farm, and I there examined twelve head of horses and mules, but only found one diseased, which was a mule five years old suffering with acute bud farcy, confined to the right hind leg. Mr. Smith had agreed to kill his two colts on the following morning; Mr. Sheppard agreed to kill his two mules, and I advised him to take the third mule over, kill her, and bury her with the others. This he did on the next day. Mr. Sheppard thinks the mules contracted the disease from the colts, and Mr. Smith believes that the colts took it from the mules. As near as I could learn, both parties had suspicious animals on their respective farms during the previous year.

On the 25th we drove eight miles southeast, to the residence of Mr. Robert A. Payne. He purchased two horses from Mr. Sheppard on the 1st day of September. One is a black gelding, five years of age, and the other a bay mare, four years old. The black horse was discharging a little at the nose at the time of purchase. On examination, I discovered two small glanders ulcers, and several yellow, pointed elevations in the right nostril; the discharge from the nostril was glutinous, but not profuse. One large farcy ulcer was situated on the side of the chest behind the right elbow, and slight induration of the right submaxillary lymphatics was present. The bay mare had a characteristic farcy ulcer above the fetlock joint on the hind leg, and several farcy tumors appearing on the thigh, shoulder, and flank. She had no appreciable enlargement of the submaxillary lymphatics, nor discharge from the nose, or other evidence of nasal affection. Mr. Payne was not at home, but I informed his wife of the nature of the disease, with the request to tell her husband that I advised the immediate destruction of both of the horses. I did not see Mr. Payne; but Mr. Sheppard told me a few days later that Payne said he could not lose the horses but must take his chances on them, as he had just purchased them for $265, and had given his note with security for the amount; that it was impossible for him to kill them at present, as he had nothing else to depend upon for the support of his family, wife and four children. We returned to town, and in the afternoon I examined a number of horses and mules, but found none diseased with glanders.

On the 27th, accompanied by Mr. Ireland, we drove eight miles southwest, to the residence of Mr. Anderson Turk. There I examined a three-year-old gray mare, and found thickening and softening with denudation of the epithelium of the nasal mucous membranes of both nostrils; a white, flaky discharge, more or less glutinous, sticking around the nasal orifice; a pale and gray, or rather leaden color, in patches on the nasal mucous surface; an external swelling over the right superior maxillary sinus, extending to the orbital fossa, superiorly and inferiorly, as far as the terminus of the zygomatic ridge. No enlargement of the submaxillary glands was perceptible; no history of external ulcers. I examined nine other horses and mules at this place, and discovered no suspicious symptoms in any, with the exception of one seven-year-old sorrel horse. He was very thin in flesh, and evidently lacked a good constitution. The nasal mucous membrane in the left nostril presented a purple streaked appearance, intermixed with coppery-hued lines radiating from the inferior border of the superior turbinated bone. There also existed some tumefaction of the left submaxillary lymphatics. I pronounced this animal suspicious, and requested Mr. Turk to keep him strictly isolated for at least two months. I declared the gray mare to be affected with chronic glanders, and recommended that she be killed and buried. Mr. Turk promised to kill her. Mr. Turk traded an old broken down mule for this mare last spring; she was then affected with a nasal discharge, but the trader stated that she had not yet quite recovered from the distemper, which she had taken a short time previously. The trader, Mr. Giralaman, of Mt. Leonard, is a regular horse-trader, traveling over the country and dealing in cheap and worthless horses. It is evident that he knew more about the mare than he disclosed, or he would not have been willing to exchange a young mare worth $150 (provided she were sound) for an old broken-down mule.

On our return to town we met Mr. L. G. Norther, who owns two horses which were suspected. I stopped him and examined his horses. One was a gray gelding about eight years of age, and the other a bay gelding about the same age. The gray horse presented characteristic glanders ulcers and tubercles on the superior turbinated bone and the meatus; had a nasal discharge from both sides; the right eye was kept partially closed, and secreted a thick yellow mucus. No swelling of the submaxillary lymphatics existed. The bay horse had some discharge from the left eye, and numerous pointed tubercles on the nasal mucous membrane of the left side; a thin watery discharge, but not profuse, flowed from the left nostril, which was slightly discolored by intermixture with blood. I declared them both glandered. The gray horse was purchased from Mr. William Sparks, of Marshall, last spring; he had a nasal discharge at the time of purchase, and when being driven would blow his nose frequently, throwing out mucus mixed with pus, but Mr. Sparks said it was nothing but epizootic, and would be all right in a few days. About a month after the purchase of this animal the other one began to manifest the same symptoms. Mr. Sparks is an extensive dealer in horses and mules. Mr. Norther told me that a year ago Sparks purchased five mules from a Mr. Humes, near Malta Bend, which had ulcers on the legs and body, and were unable to stand ordinary work. Sparks purchased them at his own risk, and soon thereafter shipped them away with a lot of other mules. I believe the gray horse of Norther's to be in the chronic stage of glanders, and the bay in an early stage, when the disease has not yet affected his general health. Mr. Norther lost one mule this summer with supposed farcy. These were all the horses and mules he possessed.

On the 28th we went 8 miles east of town, to James A. Dobbins' place. On the way we met a team hauling cord-wood, and as I noticed one of the horses had a bad eye and nasal discharge, I stopped them. The team was owned by A. H. Eastin, a near neighbor of Mr. Dobbins, and former partner in the lumber business. One of the horses was a black, sixteen years of age; he had a gluey discharge from the left nostril, and an examination revealed a tumefaction and abnormal redness of the nasal mucous membrane lining the left nostril, and several small deep ulcers and miliary tubercles were visible on the nasal septum. Both eyes were somewhat affected. A seven-year-old gray horse on the off side presented no suspicious symptoms. These horses have been exposed to the Dobbins horses. We then went to Mr. Dobbins' and there examined an eighteen-year-old horse and a two-year-old colt, neither of which manifested any evidence of disease. The reason for this examination was, that Mr. Dobbins traded for a seven-year-old horse a year ago which had a nasal discharge. Since he obtained that animal he lost, first, two mules with all the evidence of glanders and farcy, then a horse died, and lately he killed two horses which became worthless, being affected with glanders. It was discovered that the diseased horse came from Keatsville, Sheridan County, where he had been pronounced glandered, and the owner had been requested to kill him, but instead of destroying him he smuggled him out of the county, and finally Mr. Dobbins got him from a man residing at or near Slater, Saline County. After harvest, Mr. Dobbins traded him off to one of his neighbors, G. M. Ryder, and there he was killed recently, by consent of the owner, at the request of a committee sent out from Miami.

From Mr. Dobbins' place we went to John A. Singleton's. His horses were frequently exposed to the Dobbins horses. I examined three head, and found one six-year-old gray mare presenting a highly-congested nasal mucous membrane of the right side, with coppery-colored streaks appearing on the inferior meatus, extending downward to the commissure. I declared her suspicious, and requested the owner to isolate her for two months. We then went to Mr. G. M. Ryder's, and found one old blind horse (which had been working with the glandered one that had been destroyed) presenting a condition of the nasal mucous membrane very much resembling those membranes when affected with purpura or scarlatina, and Mr. Ryder stated that he had several times bled at the nose. In the absence of enlarged lymphatics and ulcers on the nasal membrane or surface of the body, I did not feel warranted in pronouncing him glandered, but requested the owner to keep him isolated for the usual length of time.

During my stay in Miami and visits to suspected places, I examined 113 head of horses and mules, and discovered, as hereinbefore described, nine cases of glanders, two of farcy, and three suspicious cases. Some of the owners of this affected stock, Mr. Payne and Mr. Norther, are poor men, and will most likely resist any measure looking to the destruction or confinement of their horses. Mr. Eastin thinks so much of his old horse that he says he will not kill him until he himself can see the plain evidence of the disease, but will build a stable for him eighty rods distant from any other horses, and will keep him therein until he becomes convinced of the true nature of the disease.

I made out a brief description of the diseased animals, recommending their speedy destruction, and also that the hitching-posts and rails in and around the city of Miami be removed. This I left with the secretary of the Anti-Horse-Thief Association, as they were to hold their regular meeting on the 1st day of November, and expected to arrange some plan whereby they can rid the community of the diseased animals.

Respectfully submitted.

M. R. TRUMBOWER,
Veterinary Inspector.

STERLING, ILL., *November* 1, 1884.

SOUTHERN CATTLE FEVER IN VIRGINIA.

During the latter part of August last, application was made to this Department by citizens of Orange County, Virginia, for a competent veterinarian to visit that locality and examine into the nature and cause of a disease which was causing the destruction of a great many cattle. Dr. W. H. Rose, of the Veterinary Experimental Station, was instructed to visit the locality and, after a thorough examination, to give such directions as he might deem best for the suppression or prevention of the further spread of the disease, should it prove to be of a contagious character. On the 28th of August Dr. Rose submitted the following report of the results of his investigations as to the nature and cause of this outbreak:

On the 26th day of August, in compliance with your instructions, I visited Orange County, Virginia, with the expectation of finding some sick cattle, but failed to do so owing to the fact that all the animals had died before my arrival. However, I found a cow which had died of the disease, that had belonged to the proprietor of the Orange Hotel. Judging from the symptoms given by Mr. Green, who treated the cow for red-water, and the *post-mortem* lesions which I found, I pronounced the disease Texas or Southern cattle fever. The connective tissues presented a deep yellow color. The spleen was enlarged, and upon section its internal structure resembled a semi-plastic mass of destroyed tissue commingled with very dark-colored blood. The liver was also enlarged, and upon section revealed a degenerated condition of its inner structure. The central portion was of a yellow color, and its cortical portion very dark in color and congested. The gall-bladder was over-distended with bile of a dark color and thickened. I also found the third stomach impacted with hardened food, and almost dry, with its mucous membrane of a dark lead color. The fourth stomach showed vascular spots on its inner and outer coverings, and the peritoneum in different parts of the alimentary tract gave evidence of more or less vascularity. At least one gallon of dark red-colored fluid escaped from the peritoneal cavity during the *post-mortem*. I could not judge the contents of the bladder on account of its lacerated condition, for the animal had been thrown from a railroad track and killed. Its posterior parts were badly mangled. The cow had been allowed to run at large in a listless condition during her illness. A butcher who opened some of the dead animals found a like condition of the stomachs, but he did not examine the other animals.

The history of this outbreak is about as follows: Captain Wallace, of Orange Court-House, brought a large drove of cattle from Charlotte, N. C., in May last, into this locality. Since that time several of the inhabitants have lost native cows, but none of the North Carolina cattle have shown symptoms of disease. Mr. W. W. Burgess, an attorney of this place, states that all the native cows affected either crossed the trail or came in direct contact with the North Carolina animals. The symptoms of those affected were listlessness, lowered heads, fever, bloody urine, constipation, a staggering gait, and a tenderness over the loins. They had fair appetites, and continued to ruminate until a late period of the disease.

Captain Wallace's herd was separated and sent to different parts of Orange County, as the following will show: Dr. E. P. Taliaferro, of Orange Court-House, has a number of these animals at pasture on his fields. Four of his native cattle have sickened and died, and one has recovered. P. H. Fry, situated some distance from the above-named place, has lost two cows. They crossed the trail of the North Carolina animals. Mr. Duncan, a neighbor, lost one cow from the same cause. W. B. Willis, of this locality, has lost several. William B. Daniels lost one, as also did a Mr. Stevens. A lot of the cattle were sent to Mitchell's Station, ten miles north of Orange Court-house. Here five native animals have died. R. C. Sale, of Crenshaw, and R. P. Groves, of Pomunkey, are both reported as having lost animals by the disease.

The Department having been notified of a second outbreak of the disease in the same locality, I returned to Orange County on Monday, September 1. I found the affected cattle at Mitchell's Station, situated about ten miles north of Orange Court-House, on the Midland Railroad. The outbreak occurred here among cattle belonging to Mr. James B. Lewis. This gentleman had purchased of Captain Wallace, in May last, about eighty head of Charlotte (N. C.) cattle from the herd above alluded to, and had placed them in the pasture with his native animals. Since their arrival seven of his native cows had sickened and died. One cow had partly recovered, but remains in a very poor condition. The ninth native animal shows symptoms of disease (temperature 106⅗° Fah.), and I think she will die. Three milch cows belonging to other parties have died on this place, making a total of ten deaths up to date, with the prospect of another before to-morrow. In one of the pasture-fields I found seven calves in apparently good health. Mr. Lewis seemed surprised to find them so well, as four of them had suckled their sick dams as long as they could obtain milk.

I made a *post-mortem* examination of a dead cow which had been hauled from the barn-yard to-day, and found the usual appearance of the spleen, liver, and kidneys as found in all cases of Southern cattle fever.

As Dr. Summerville reported this outbreak to the Department, I concluded to visit his place. He lives one mile north of the Lewis farm, and is the owner of a great many cattle. With the exception of a yoke of oxen none of his animals had been infected. They had been driven over a road where the Charlotte cattle had previously passed. I found one of them very sick (temperature 107⅘° Fah.), and in a recumbent position, with its muzzle resting upon the ground.

Mr. George L. Berry, who lives three miles from the Lewis farm, lost five cows during the month of August. He stated that he had had no Charlotte cattle on his place, but acknowledged that he last fall purchased cattle from Pittsylvania County, Virginia, which were still on his place.

A Mr. Thomas, who lives at Mitchell's Station, has one sick cow, which was infected by mingling with the new arrivals.

During my investigations here I was informed of other outbreaks of disease in different parts of this and in other counties, and I concluded to follow the trail of all the Charlotte cattle that had been sold and distributed by Captain Wallace. On the second day of September I examined cattle belonging to Mr. R. C. Sale, who lives eight miles southeast of Mitchell's Station. Forty head of the Wallace cattle are on this farm, but none of the native stock have as yet shown symptoms of the disease.

Mr. J. T. Grasty, a relative of Mr. Sale, owns the adjoining farm. He has eighty head of the same stock mixed with his native animals, but no serious results have occurred among them up to the present time. It may be that these cattle are free from infection, as Captain Wallace brought in two lots, one in May and the other in the month of June.

I found no change among the cattle at Orange Court-House since my former visit. I heard here that cattle were dying of the disease at Charlottesville, Albemarle County, but on visiting that place I learned from Messrs. C. H. and W. R. Burnley that such was not the case. These gentlemen conduct large monthly sales of cattle, but they will not admit animals from the permanently infected district into their sales during the hot months. They have postponed some sales for fear of infection. Mr. Burnley stated that Captain Wallace had offered him some of the Charlotte cattle, but he refused to risk such stock in the market. The report of illness among cattle in this locality no doubt arose from the death by black quarter of five or six calves.

Mr. Willis, a druggist of Charlottesville, who owns that portion of the pasture-fields which became infected from the Halifax cattle that were grazed on the Jeffrey place last year, informed me that many native cattle have pastured on the place this year without any ill effects.

On my return to Washington I found a large number of Charlotte cattle at Rapidan Station, on the Midland Railroad, a short distance from Mitchell's. They belonged to Mr. Alexander, who claims that none of his native cattle have been infected.

FATAL OUTBREAK OF SOUTHERN CATTLE FEVER.

Hon. George B. Loring,
Commissioner of Agriculture:

Sir: I have the honor to report a serious outbreak of Texas or Southern cattle fever at what is known as the Deerfield Stock Farm, Spencer Island, Butler County, Kansas. So far as I have been able to collect the facts in the case, they are as follows:

On the 18th of August last, Messrs. H. F. & J. C. Robertson, of Sonora, Muskingum County, Ohio, left that place with twenty-one head of thorough-bred short-horn cattle. Six of them were valuable bulls; the remainder consisted of cows and one and two year old heifers. From Sonora they passed over the Baltimore and Ohio Railroad to Chicago, and thence over the Wabash, Saint Louis and Pacific Railroad to Hannibal, Mo. At Hannibal the Messrs. Robertson requested the railroad company to allow them to unload the cattle for rest and feed at some small station west of Hannibal, as they feared the stock-yards at this place might in some way be infected. This request was refused, and they were assured that no case of Texas fever had been known in these yards. Accordingly, all of the cattle except six bulls were unloaded on the morning of the 22d of August in good condition. As an extra precaution, the six bulls were watered and fed in the car; in fact, were not out of the car from the time they were loaded in Ohio until they arrived at their destination in this State. They remained in Hannibal some twelve or fifteen hours, leaving there on the evening of the 22d of August and arriving in Kansas City on the following evening. They left Kansas City the same night, passing over the Atchison, Topeka and Santa Fé Railroad to Florence, Kans., where they arrived the evening of August 24. Here they were unloaded (the cows that night, the bulls the next morning), and were driven to their present location, Spencer Island, Butler County, Kansas.

On the 3d day of September, just twelve days after their confinement in the stock-yards at Hannibal, two of the most valuable cows were taken sick with Texas fever. Of these one died on the 5th and the other on the 7th. On the 4th six others, also cows or heifers, were affected. One of these died on the 6th and two on the 7th. Two more heifers were taken on the 5th, one of which died on the 8th, and the other one is expected to die to-day, September 12.

Of those attacked on the 4th, one has, to all appearances, entirely recovered, and the chances seem to be in favor of two others. Thus, of the ten animals attacked, seven are dead, one recovered, and two convalescent. The Messrs. Robertson place their loss at about $1,500.

As soon as the disease made its appearance, the whole herd, both sick and well, were treated with aconite, Epsom salts, belladonna, &c.

It is a matter worthy of particular attention that none of the bulls were troubled in the least, a fact which the Messrs. Robertson explain as being due to their not having been unloaded at Hannibal. This point is a resting place for Texas cattle on their way to Chicago. There were nine car-loads there at the time the cattle above referred to were unloaded.

There seems to be no room for doubt that the cattle in this case were infected in the stock-yards at Hannibal. The only effectual remedy that suggests itself is to have pens separate and apart for the exclusive use of Texas cattle, and never allow them to be unloaded in pens used for the accommodation of other cattle, except during months when sharp frosts are experienced. I have written to the superintendent of the stock-yards at Hannibal, calling his attention to this case, and also to the circular of the Department of Agriculture in regard to the disease, and making to him the suggestion that separate pens be used, as above recommended.

I am, very respectfully, yours,

J. H. FULLINWIDER,
Inspector, Bureau of Animal Industry.

EL DORADO, KANS., *September* 12, 1884.

PARASITIC BRONCHITIS AMONG CALVES.

Hon. **George B. Loring**,
Commissioner of Agriculture:

Sir: I received your telegram of the 14th, ordering me to go to Alpha, Mo., and there to examine some calves belonging to Mr. O. L. Casebeer. I left home on the morning of the 17th, and arrived at Trenton, Mo., (the nearest railway station to Alpha), on the evening of the 18th. On the 19th I hired a livery team and driver and proceeded to Alpha, Grundy County, a distance of fourteen miles; from thence I went one mile south, to the residence of Mr. Casebeer. He purchased, on the 10th day of September, 10 head of spring calves from Mr. E. C. Gregg, near Wheeler, Livingston County, and on the same day 22 head from Mr. Jasper Fell, of the same place. These calves appeared thin and rough-haired when purchased, but having been recently shipped from Western New York and from Ohio, it was supposed to be due to a ten days' journey on the cars. When Casebeer drove these calves home, a distance of fifteen miles, he observed that a number of them were afflicted with a cough. This cough afterwards became more frequent, the breathing labored and painful, and rapid emaciation set in. Soon the disease spread through the whole herd; even two calves and five cows of his own raising became affected. On the 13th of October one of the imported calves died, a few days later a second one, then a third, and a fourth one on the 16th of November. In the mean time the disease was communicated to a number of calves in an adjoining field belonging to Mr. Lemuel Spence.

I examined the remaining calves belonging to Mr. Casebeer, and discovered a severe bronchial catarrh, mucus being discharged from the nose, rattling in the trachea in inspiration and expiration, strong vesicular murmurs in both lungs, and excessive coughing when they were made to run. The animal which died on the 16th had not been buried, and although it was so far advanced in decomposition that it was unfit to handle, nevertheless I cut open the thorax and trachea, and discovered a large number of lung worms (*Strongylus micrurus*) in the trachea and large bronchi. As all the symptoms of disease present in the herd, as well as the history, corresponded with the disease known as parasitic bronchitis, I informed the owner of the nature, cause, &c., and recommended the continuance of fumigation with burning sulphur, which he had previously been using. I also prescribed for internal administration oleum terebinthinæ in oleum lini, and a powder composed of ferri sulphas, gentian, zingiber, and capsicum.

From Casebeer's place I drove to that of Mr. Spence, and was informed by this gentleman that he had lost 2 out of 27 head of calves, but all of them were affected with more or less of a cough. These calves were, during the months of September and October, pastured in a field next to Casebeer's cattle, and would occasionally slip through a willow hedge which divided the two herds and mingle with the Casebeer cattle.

Having been informed that Messrs. Fell and Gregg had lost a number of calves, I thought it advisable for me to visit their herds. On the morning of the 20th, I drove from Alpha to Mr. Gregg's farm, which is located four miles north of Wheeler, and found both Mr. Gregg and Mr. Fell at home. Fell resides on the opposite side of the road from Gregg. Here I obtained the following history: In the early part of the month of August, Mr. Jasper Fell purchased, in Chautauqua County, New York, and in Warren County, Pennsylvania, 105 head of calves. They were collected from farmers and dairy stables. He shipped them at Columbus, Pa., and brought them to Cincinnati, Ohio. At the latter place he added 177 head, purchased by Mr. Gregg in Trumbull County, Ohio, and 2 others they purchased at Cincinnati, which had been brought to the stock-yards for sale by a farmer. They reshipped and arrived at Wheeler, Mo., on the 19th of August with 284 head. They divided the herd into two equal numbers (142) and took them to their farms. In the middle or towards the latter part of September the calves began to cough, and Mr. Gregg lost 21 out of 119, including 2 which I killed for examination. He had, however, sold 10 head to Mr. Casebeer and 13 to Mr. Real. Real purchased his on the 29th of August, and had not suffered any loss when last heard from. Gregg had 14 head of native cattle, all of which are also coughing. Mr. Fell sold 22 head to Casebeer, and has lost from the balance of his herd (120) 18 head.

All of the Gregg and Fell calves are affected, and a large number will yet die with the disease, as many of them are greatly reduced in flesh, very weak, and the cough gradually becoming more painful and weak. They have been dosed with saltpeter and sulphur and fumigated with sulphur for several weeks. The sulphur fumigations have been used to excess, both in frequency and strength. In short, they are as miserable a lot of calves as I have ever seen, coughing almost incessantly, almost too weak to walk; coughing up and discharging worms, pus, mucus, and, in some instances, blood from the nostrils. I killed two for examination; one of them had been suffering about two weeks; was lying on a bunch of hay in the stable-yard, from which it had been unable to arise for three days; reduced to a skeleton; breathing very shallow and laborious, and appetite altogether absent. On examination of the lungs I found red hepatization, atelectasis, vesicular emphysema, small abscesses (which might easily be mistaken for tuberculous deposits) in many parts of the lungs, more especially towards the free border. On section many of the bronchial tubes were found to be occluded with *Strongyli*, reaching from the bifurcation of the trachea to the smallest divisions. The lungs contained no less than one pint of these worms, *Strongylus micrurus*. The muscular structure of the animal was blanched, anæmic, and softened. The fourth stomach contained quite a number of *Strongylus contortus*, but not in sufficient numbers to affect digestion. I wish to say here that this is the first calf in which I have found these entozoa. The cæcum contained about two dozen whip-worms (*Trichocephalus affinis*). The second calf which we killed presented similar conditions, embracing the three classes of entozoa. This was one of the first that began to cough, and a great amount of lung tissue was congested and filled with small abscesses. It is possible that the latter were worm nests, but as I am not a microscopist I was unable to determine the exact nature of those abscesses. It is strange, however, that in both cases, while there was considerable red hepatization and evident inflammatory action in the lung structure, still the localized pneumonitis did not progress toward destruction of tissue, as would be expected, but appeared to remain in a quiescent state, without resorption or degeneration to any greater extent than was shown by the small abscesses. I recommended the same remedies to Gregg and Fell as I had to Casebeer.

Had I known that you would not need my services soon in other places I would have remained longer, and experimented with inhalation of chloroform and carbolic acid or sulphophenol. I have little faith in the sulphurous anhydride, in rue, savin, tar smoke, turpentine, or any of the remedies which are so universally recommended in our veterinary literature. I have tried the chloroform and carbolic acid once, and believe that I obtained excellent results from it. As the acid is a great germicide, and as it can certainly be directly conveyed into the lungs by the vapor of chloroform, I have faith to believe that it will yield good results, provided it is carefully administered. The inhalations should be largely diluted with air, so that the irritant effect of the medicament may not injure the already hyperæmic mucosæ.

Respectfully submitted.

M. R. TRUMBOWER,
Veterinary Inspector.

STERLING, ILL., *November* 29, 1884.

HISTORY OF LUNG PLAGUE IN RICHMOND COUNTY, NEW YORK.

Hon. GEO. B. LORING,
Commissioner of Agriculture:

SIR: My investigations have enabled me to give the following history of the invasion and prevalence of contagious pleuro-pneumonia in Richmond County, New York:

In December, 1876, Mrs. McIntyre owned a herd of 10 cows. About the first of January of the following year she bought 4 more of a dealer in New York City. These 4 cows sickened and died, as did 4 of the original herd, making 8 in all. In the early part of February, 1877, cows from this infected stable were sent to the stable of R. W. Cameron. Mr. Cameron's herd contained 2 bulls and 30 cows—imported Ayrshires. About the middle of March, 2 of his animals showed symptoms of lung plague, and on the 28th of the same month both died. I held a *post-mortem* on these animals, and pronounced the disease contagious pleuro-pneumonia. Mr. Cameron was unwilling to accept my diagnosis, so I forwarded a section of the hepatized lung to Dr. James Law, accompanied with a history of the case. He pronounced it a decided case of lung plague. Other animals sickened, and by the middle of May he had lost 14 cows, 1 bull, and 1 calf, making 16, or 24 in all.

Mrs. Brady, a neighbor of Mrs. McIntyre, lost 3 animals with the disease in the latter part of May of this year. About the same time Mrs. Flake lost 3 cows.

John Clifford owned a herd of 17 cows. In the spring of 1878 he purchased 3 animals from a New York dealer, and in about six weeks afterward the 3 cows died. Some of the animals in his original herd were soon taken sick, and 4 of them died, making a total loss of 7. He traded the remainder of the herd to a dealer.

About this time Mr. J. Gallaghan, a neighbor, had 12 cows attacked with the disease. Eight of these died, and the other 4 were sold to a dealer. Mrs. O'Brien, also a neighbor, lost 3 cows with lung plague. Her place is located about two miles from where the outbreak occurred in 1878.

In the spring of 1879 Jacob Mauer, of Concord, had 12 cows, and bought 2 more from a Brooklyn dealer. The latter 2 and 10 of the others soon died of contagious pleuro-pneumonia. The other 2 were sick, and were turned on the commons, where they finally died, making a total loss of 14. Mrs. Stalty had 16 cows grazing on the same commons. These animals contracted the disease, and 11 out of the 16 head died. At about the same time R. Phair had 4 cows grazing on the same pasture. They contracted the disease, and 3 out of the 4 died. Mr. Silvey, also of Concord, lost 2 cows.

In May, 1879, G. Gallaghan, of Tompkinsville, previously mentioned, had 6 cows. In company with Prof. James Law we killed 1 of these animals and quarantined the other 5. Two of these afterward died, making 3 in all.

In the spring of 1880 Mr. Wormsly had 8 cows grazing on the commons known as Van Wagner's unfenced farm. These cows were brought from New York City. Five of them were attacked with the disease and died. About the same time and place Mr. Harper lost 1; Mr. Ackerley 4; Mr. Leight 4; Mr. Licherman 3, and Mr. Doran 2.

On the first day of April of the above year, Mr. Christian Zebender, of Stapleton, owned 12 cows and 1 bull. He purchased some of these cows from a dealer in New York City. Three of them were attacked with contagious pleuro-pneumonia, and by order of Gen. M. R. Patrick I quarantined the herd. Eight of the animals died and 5 were killed by a butcher, all showing signs of lung plague. Mr. Brammer, a neighbor, lost 1 cow by the same disease.

In February, 1880, Thomas Foley had a herd of 32 cows. In this herd was one he bought from a Brooklyn dealer, which sickened and infected most of the others. By order of Gen. M. R. Patrick I quarantined the stables, but by the 11th of May he had lost 17 head. One was killed, which made the loss 18 in all. About the same time Mr. Copps, of Tompkinsville, had a herd of 15 cows, 2 of which died of lung plague. The other 13 he traded to a dealer for about $10 per head. Mr. Haas of the same place, bought some cows from a Brooklyn dealer, and soon the disease broke out in his herd of 20 cows. During the next three years he lost about 30 cows, which ruined him.

February 14, 1881, I was called to attend a sick horse belonging to the Child's Nursery Hospital. The institution kept a herd of cows for the use of four or five hundred children. I noticed a very fine looking cow standing in one corner away from the rest, and upon examination found her suffering with contagious pleuro-pneumonia. Upon inquiry I found they had purchased her from Moses Maybaum, of Linoleumville. This man has distributed more disease in this county than any other person in this locality. About a month before this time I informed Mrs. DuBoise, the head of the institution, that she had a cow in the byre afflicted with a contagious disease, and advised her to destroy or remove her from the rest. She sent for the butcher, but when he saw her he refused to kill her, saying she was the fattest and finest animal in the herd. In about a month from that time she died. Several others showing symptoms of the disease, the whole herd was sold to a Mr. Cutter, of Willow Brook, the institution agreeing to take 300 quarts of milk per day; but in May between two and three hundred children were taken sick. Dr. Harris, the secretary of the State Board of Health was notified, and he and Dr. Walser, the attending physician, called upon me, and together, on the 10th day of May, we visited the institution and this diseased herd. On examination most of the cows were found suffering with contagious pleuro-pneumonia in its worst form. Upon inquiry, we learned that 11 head had died and others were suffering with the plague. By order of General Patrick I quarantined the remainder of the herd on the farm, but Cutter soon sold them to Moses Maybaum, for $10 per head.

In May, 1881, Mr. Harris had a herd of cows on the Conner farm near the Nursery Hospital. These cows—about 16 in number—contracted the disease. Dr. Harris, of the State board of health, requested the sheriff to assist me if necessary in carrying out measures for the suppression of the plague. On our arrival at the farm we found 6 dead animals. The remaining 10 were taken away on Sunday night, together with the horses, wagons, harness, milk pails, and cans. About the same time, and near this farm, Richard Conner lost 4 animals; J. Curry lost 2, and Mr. Mead lost 2.

March 24, 1882, Mr. Sersemar, on the Manor road, had 23 cows and 1 bull. His last purchase was from a New York dealer, and this animal was the first to get sick. Four cows and the bull died. The remaining 19 head were sold. In the same neighborhood George Atler had a herd of 14 cows, 7 calves, and 1 bull. His cattle contracted the disease from cows coming from infected stables for service, and he lost his whole herd of 22 animals.

In September, 1882, Mr. Selig, one field from Mr. Atler, had a herd of 27 cows, 4 calves, and 1 bull. His last purchase was from a New York dealer, and this animal was the first one that showed symptoms of disease. I quarantined them by order of Mr. Denniston. Eight of the animals died, and the remainder were sold to a butcher by the name of Lewis.

In June, 1883, Patrick Smith, of Toadt Hill, had a herd of 15 cows. He purchased some of his cows from Moses Maybaum in the spring, and the last ones that were purchased were the first ones taken sick. Professor Law accompanied me on a visit to this herd. Four animals died previous to our visit and 2 died shortly after, making a total loss of 6. The remaining 9 animals he sold to Maybaum. Patrick Collins, adjoining this farm, lost 4 animals out of a herd of 12; Mrs. James, a neighbor to Collins, lost 2 cows and 1 calf; Mr. Bradley, of Willow Brook, lost 3 animals; Mr. Layton, in the same neighborhood, lost 11 head. Margaret Bean, of Willow Brook, close by, had 4 cows attacked with the plague. She sold them to Moses Maybaum, of whom she purchased the last cow.

Mr. Drake, living on the Clove road, had a herd of 20 cows. Some of the animals showing symptoms of the disease, he sold the entire herd. Charles Janin had 10 cows in the adjoining field; 4 died and he sold the remainder; Mr. Egbert, of Northfield, lost 8; Mr. Morris, of the same place, lost 10; Mr. McQuade, of Castleton, lost 4; Mr. McSorley lost 2; Mrs. Davis lost 2; Mrs. Carley lost 2; Mrs. McAvoy lost 2; Mrs. Dugan lost 1, and Mr. Suter, of the same place, lost 2 and sold 3.

They generally keep about 20 cows and 1 bull at Sailor's Snug Harbor. These animals were grazed on a field adjoining Mr. Suter's, and contracted the disease from his cows. Animals in this herd continued to die one or two at a time from July, 1883, to February, 1884, at which time 8 had died and 4 were sick. On April 10th following, the 4 sick animals were shot, making a total loss of 12 cows.

A Mr. Emmons and a Mr. Herpeck lost 2 animals each.

In addition to the above, the following-named persons have lost, during the current year, the number of animals set opposite their names by contagious pleuro-pneumonia, viz:

Name.	No.	Name.	No.
Mrs. McArney	1	Mr. Castle	6
Mr. Schmeizer	1	Mr. Gundecker	1
Mr. Dwyer	1	Mr. Mead	1
Thomas Dunn	3	Mr. Zipp	2
John Drummond	1	Mr. Lawrence	5
George Whitford	1	Mrs. Lew	2
Mr. Burk	1	Mrs. Lastrange	2
Mr. Clingman	1		
		Total	29

The above gives a grand total of 351 animals lost by contagious pleuro-pneumonia in this county during the past eight years.

Respectfully submitted.

WILLIAM ROSE,
Veterinary Inspector.

RICHMOND COUNTY, N. Y., *October* 31, 1884.

UNITED STATES CATTLE QUARANTINE.

Superintendents of the United States cattle quarantine stations report the number and breed of each lot of cattle received from July 1, 1884, to January 1, 1885, the port of shipment, date of arrival; also the importer's or consignee's name and post-office address, as follows:

PATAPSCO STATION, NEAR BALTIMORE, MARYLAND.

[*Dr. Alvord H. Rose, Superintendent.*]

Date of arrival.	Port of shipment.	Name of breed.	No. received.	Importer's name.	Post-office address.
1884.					
July 10	Liverpool	Jersey	25	H. R. Tucker & Co.	Baltimore, Md.
Aug. 12	do	Hereford	50	W. P. Herring	Kansas City, Mo.
Dec. 3	do	do	8	George Aston	Elyria, Ohio.

There were no arrivals at this station during September, October, or November.

COOPERSBURG STATION, PENNSYLVANIA.

[*Dr. Francis Bridge, Superintendent.*]

Date of arrival.	Port of shipment.	Name of breed.	No. received.	Importer's name.	Post-office address.
1884.					
Sept. 25	Southampton	Jersey	98	T. S. Cooper	Coopersburg, Pa.
25	do	Guernsey	72	J. W. Fuller	Catasauqua, Pa.

GARFIELD STATION, NEW JERSEY.

[*Dr. A. M. Farrington, Superintendent.*]

Date of arrival.	Port of shipment.	Name of breed.	No. received.	Importer's name.	Post-office address.
1884.					
July 16	London	Holstein	5	W. C. Dorwin	Owego, N. Y.
26	Antwerp	do	96	Wm. Koch	Staten Island, N. Y.
Aug. 2	London	Hereford	20	James A. Perry	Wilmington, Ill.
9	do	Holstein	43	T. F. Koch	San Francisco, Cal.
9	do	do	50	George L. Wells	Weathersfield, Conn.
30	do	do	76	Guthrie & Bell	Louisville, Ky.
30	do	do	18	G. W. Thompson	Stelton, N. J.
Sept. 9	Bremen	do	7	H. Stelloh	Milwaukee, Wis.
20	Amsterdam	do	50	Marsh & Seeley	N. Milford, Conn.
29	Antwerp	do	34	E. Hindekoper	Meadville, Pa.
Oct. 4	Bristol	Jersey	8	R. M. Hoe	New York City, N. Y.
10	Antwerp	Holstein	24	S. P. Dyt	Passaic, N. J.
13	London	Sussex	11	Overton Lea	Nashville, Tenn.
29	do	Jersey	11	J. F. Maxfield	Bloomfield, N. J.
29	do	do	64	Wm. Simpson	New York City, N. Y.
Nov. 24	do	Holstein	20	W. L. Breese	Do.
Dec. 30	do	Galloway	13	F. G. Babcock	Hornellsville, N. Y.
30	Rotterdam	Holstein	58	Sluiter Bros	Garfield, N. J.
13	do	do	80	Albert French	Cincinnati, Ohio.

WALTHAM STATION, MASSACHUSETTS.

[*Dr. Madison Bunker, Superintendent.*]

Date of arrival.	Port of shipment.	Name of breed.	No. received.	Importer's name.	Post-office address.
1884.					
July 5	Amsterdam	Holstein	107	W. A. Russell	Lawrence, Mass.
Aug. 27	Fayal	Grade	1	C. L. Bartlett & Co.	Boston, Mass.
Sept. 2	Amsterdam	Holstein	169	Smiths & Powell	Syracuse, N. Y.
2	London	Shorthorn	2	F. Brigham & Co	Boston, Mass.
14	Amsterdam	Holstein	118	S. S. Mann & Co.	Elgin, Ill.
14	....do	....do	68	Bidwell & Perry	Monterey, Mass.
23	Antwerp	....do	80	C. W. Wolcott	Readville, Mass.
Oct. 6	Amsterdam	....do	85	D. H. Archibald	Oxford, Miss.
9	....do	....do	108	F. C. Stevens	Attica, N. Y.
20	....do	....do	150	H. Langworthy	W. Edmeston, N. Y.
20	....do	....do	53	H. E. Boardman	Rochester, N. Y.
Nov. 8	....do	....do	40	E. M. Washburn	Lenox, Mass.
8	Glasgow	Angus	8	Waldo Bros.	Boston, Mass.
22	....do	Galloway	92	Cunningham & Co.	Glasgow, Scotland.

Whole number of cattle received at the quarantine stations from July 1, 1884, to January 1, 1885, as given in detail above.

Patapsco Station	78
Coopersburg Station	165
Garfield Station	633
Waltham Station	976
Total	1,852

As the necessity for these stations will be permanent, it would seem advisable for the Government to acquire more secure possession, either by long lease or purchase, of suitable grounds, which could be placed in a satisfactory condition for cleaning and disinfection. While not entirely free from objections, the present arrangements must be considered as very satisfactory when we take into account their temporary character, the small appropriations for establishing them, and the short time during which they have been in operation.

BUREAU OF ANIMAL INDUSTRY.

The following is the text of the act passed during the last session of Congress for the establishment of a Bureau of Animal Industry:

Be it enacted by the Senate and House of Representatives of the United States of America in Congress assembled, That the Commissioner of Agriculture shall organize in his Department a Bureau of Animal Industry, and shall appoint a Chief thereof, who shall be a competent veterinary surgeon, and whose duty it shall be to investigate and report upon the condition of the domestic animals of the United States, their protection and use, and also inquire into and report the causes of contagious, infectious, and communicable diseases among them, and the means for the prevention and cure of the same, and to collect such information on these subjects as shall be valuable to the agricultural and commercial interests of the country; and the Commissioner of Agriculture is hereby authorized to employ a force sufficient for this purpose, not to exceed twenty persons at any one time. The salary of the Chief of said Bureau shall be three thousand dollars per annum; and the Commissioner shall appoint a clerk for said Bureau, with a salary of one thousand five hundred dollars per annum.

SEC. 2. That the Commissioner of Agriculture is authorized to appoint two competent agents, who shall be practical stock-raisers or experienced business men familiar with questions pertaining to commercial transactions in live stock, whose duty it shall be, under the instructions of the Commissioner of Agriculture, to examine and report upon the best methods of treating, transporting, and caring for animals, and the means to be adopted for the suppression and extirpation of contagious pleuro-pneumonia, and to provide against the spread of other dangerous contagious, infectious, and communicable diseases. The compensation of said agents shall be at the rate of ten dollars per diem, with all necessary expenses, while engaged in the actual performance of their duties under this act, when absent from their usual place of business or residence as such agent.

SEC. 3. That it shall be the duty of the Commissioner of Agriculture to prepare such rules and regulations as he may deem necessary for the speedy and effectual suppression and extirpation of said diseases, and to certify such rules and regulations to the executive authority of each State and Territory, and invite said authorities to co-operate in the execution and enforcement of this act. Whenever the plans and methods of the Commissioner of Agriculture shall be accepted by any State or Territory in which pleuro-pneumonia or other contagious, infectious, or communicable disease is declared to exist, or such State or Territory shall have adopted plans and methods for the suppression and extirpation of said diseases, and such plans and methods shall be accepted by the Commissioner of Agriculture, and whenever the governor of a State or other properly constituted authorities signify their readiness to co-operate for the extinction of any contagious, infectious, or communicable disease in conformity with the provisions of this act, the Commissioner of Agriculture is hereby authorized to expend so much of the money appropriated by this act as may be necessary in such investigations, and in such disinfection and quarantine measures as may be necessary to prevent the spread of the disease from one State or Territory into another.

SEC. 4. That in order to promote the exportation of live stock from the United States the Commissioner of Agriculture shall make special investigation as to the existence of pleuro-pneumonia, or any contagious, infectious, or communicable disease, along the dividing lines between the United States and foreign countries, and along the lines of transportation from all parts of the United States to ports from which live stock are exported, and make report of the results of such investigation to the Secretary of the Treasury, who shall, from time to time, establish such regulations concerning the exportation and transportation of live stock as the results of said investigations may require.

SEC. 5. That to prevent the exportation from any port of the United States to any port in a foreign country of live stock affected with any contagious, infectious, or communicable disease, and especially pleuro-pneumonia, the Secretary of the Treasury be, and he is hereby, authorized to take such steps and adopt such measures, not inconsistent with the provisions of this act, as he may deem necessary.

SEC. 6. That no railroad company within the United States, or the owners or masters of any steam or sailing or other vessel or boat, shall receive for transportation or transport, from one State or Territory to another, or from any State into the District of Columbia, or from the District into any State, any live stock affected with any contagious, infectious, or communicable disease, and especially the disease known as pleuro-pneumonia; nor shall any person, company, or corporation deliver for such transportation to any railroad company, or master or owner of any boat or vessel, any live stock, knowing them to be affected with any contagious, infectious, or communicable disease; nor shall any person, company, or corporation drive on foot or transport in private conveyance from one State or Territory to another, or from any State into the District of Columbia, or from the District into any State, any live stock, knowing them to be affected with any contagious, infectious, or communicable disease, and especially the disease known as pleuro-pneumonia: *Provided*, That the so-called splenetic or Texas fever shall not be considered a contagious, infectious or communicable disease within the meaning of sections four, five, six and seven of this act, as to cattle being transported by rail to market for slaughter, when the same are unloaded only to be fed and watered in lots on the way thereto.

SEC. 7. That it shall be the duty of the Commissioner of Agriculture to notify, in writing, the proper officials or agents of any railroad, steamboat, or other transportation company doing business in or through any infected locality, and by publication in such newspapers as he may select, of the existence of said contagion; and any person or persons operating any such railroad, or master or owner of any boat or vessel, or owner or custodian of or person having control over such cattle or other live stock within such infected district, who shall knowingly violate the provisions of section six of this act, shall be guilty of a misdemeanor, and, upon conviction, shall be punished by a fine of not less than one hundred nor more than five thousand dollars, or by imprisonment for not more that one year, or by both such fine and imprisonment.

SEC. 8. That whenever any contagious, infectious, or communicable disease affecting domestic animals, and especially the disease known as pleuro-pneumonia, shall be brought into or shall break out in the District of Columbia, it shall be the duty of the Commissioners of said District to take measures to suppress the same promptly and to prevent the same from spreading; and for this purpose the said Commissioners are hereby empowered to order and require that any premises, farm, or farms where such disease exists, or has existed, be put in quarantine; to order all or any animals coming into the District to be detained at any place or places for the purpose of nspection and examination; to prescribe regulations for and to require the destruction of animals affected with contagious, infectious, or communicable disease, and for the proper disposition of their hides and carcasses; to prescribe regulations for disinfection, and such other regulations as they may deem necessary to prevent infection or contagion being communicated, and shall report to the Commissioner of Agriculture whatever they may do in pursuance of the provisions of this section.

SEC. 9. That it shall be the duty of the several United States district attorneys to prosecute all violations of this act which shall be brought to their notice or knowledge by any person making the complaint under oath; and the same shall be heard before any district or circuit court of the United States or Territorial court holden within the district in which the violation of this act has been committed.

SEC. 10. That the sum of one hundred and fifty thousand dollars, to be immediately available, or so much thereof as may be necessary, is hereby appropriated, out of any moneys in the Treasury not otherwise appropriated, to carry into effect the provisions of this act.

SEC. 11. That the Commissioner of Agriculture shall report annually to Congress, at the commencement of each session, a list of the names of all persons employed, an itemized statement of all expenditures under this act, and full particulars of the means adopted and carried into effect for the suppression of contagious, infectious, or communicable diseases among domestic animals.

Approved, May 29, 1884.

TRICHINIASIS.

[The following article on Trichinæ and Trichiniasis was contributed by Dr. D. E. Salmon, Chief of the Bureau of Animal Industry, to the report of the Commission, of which he was a member, appointed by the President to investigate the condition of the swine industry, and the pork product of the United States. It embraces the researches which have been made in regard to this subject up to this time.]

EXTENT OF TRICHINIASIS IN AMERICA AND EUROPE.

This subject being, in the present attitude of certain foreign Governments in regard to American pork products, the most important of all the questions that have received our attention, we have given it a very careful consideration. The alleged frequency of trichiniasis in American hogs has been *the* reason insisted upon by the various countries which have prohibited the importation of such products; for, while it is true that other objections have been advanced, particularly in France, none of these have sufficient foundation in fact to stand the test of even a superficial examination. It is, however, not a question of the prevalence of trichiniasis here and its absence in other countries, since this parasite has been found infecting the hogs and other flesh-eating animals in the most widely separated portions of the earth. Dr. Manson examined 225 specimens of Chinese pork and found 2 or nearly 1 per cent. infected.[*] Dr. Wartable has described epidemics near the sources of the Jordan resulting from eating the flesh of the wild boar,[†] and in every European country in which inspections have been made a very considerable proportion of trichinous animals have been discovered.

Certain writers have pretended that the animals of France have never been affected with trichiniasis,[‡] but this conclusion seems to have been reached without any investigations. A large proportion of the rats of Paris were long since found to be infected,[§] and in 1879 a serious epidemic of trichiniasis, known as that of Crépy-en-Valois, occurred, in which sixteen persons sickened from eating the flesh of a native animal.[‖]

That trichinæ also exist in America and infest a small proportion of American hogs is a fact that must be admitted, but it is a more difficult matter to compare the frequency of American and European infection than has usually been supposed. American hogs have usually been examined by microscopists who were competent to do the work and who would not overlook a single case, while in Germany there has been an immense number of inspectors employed (18,581 in 1881), many of whom were utterly incompetent. An examination in 1877 showed that many of the microscopes were useless, that glasses used were too dirty to permit the examination, and that some of the inspectors were incapable of detecting the parasite.[¶] Even as late as 1881 there were complaints in regard to the incompetency of inspectors, and the continued recurrence of trichiniasis among people from eating inspected meats demonstrates that these complaints were not made without reason.* Even the German inspections of American meats cannot be taken as a fair comparison with the average of their inspections of indigenous animals, for the reason that our meats are examined in their larger cities and by their most competent inspectors. It is absolutely necessary to bear these facts in mind, in considering the figures which are given as representing the results of the microscopic examination of American and European pork.

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[*] Imp. Customs. Med. Report, Shanghai, XXI (1881), p. 26.
[†] Lancet, August 4, 1883.
[‡] J. Chatin: La trichine et la trichinose.
[§] Davaine: Traité des entozoaires, &c., p. 755.
[‖] Gazette des Hôpitaux, February 20, 1879.
[¶] Vierteljahrschrift f. Ger., Med., &c., N. F. XXX, p. 175–181.

PROPORTION OF AMERICAN HOGS INFECTED WITH TRICHINIASIS.

The hogs in Dearborn County, Indiana, seem to be infected in a larger proportion than anywhere else in the country, or at least were in 1874. Drs. Harding and Robbins examined 245 animals slaughtered near Lawrenceburg, and found that 40, or 16⅓ per cent., contained this parasite. This seems to be far beyond the average, however, even in this center of infection, for Drs. Gatch and Miller examined 200 animals at the same place and only found 13 infected, or 6.5 per cent.†

In 1866 Belfield and Atwood are reported to have found 2 per cent. of the hogs slaughtered in Chicago infected, and in 1878 an examination of 100 animals at the same place indicated that 8 per cent. contained trichinæ.

From 1879 to 1881 Dr. F. S. Billings, of Boston, examined 8,773 hogs, of which 347, or 4 per cent., were reported as containing trichinæ.

Dr. Deveron, of New Orleans, inspected 5,400 hogs in 1881, of which only 22, or 0.4 per cent., were trichinous.‡ Of these animals 529 came from Saint Louis, and among them were 18 infected ones, being 3.4 per cent.; 241 came from Louisville, and 2 of these, or 0.83 per cent., contained trichinæ; 484 from unknown parts of the West, had but 2 infected, or 0.4 per cent., while the remaining 4,146, mostly from the South, were free from this parasite.

Dr. C. A. Simpson examined 30 hogs at Atlanta, which were mostly from Tennessee, without finding any infected, and Dr. R. W. Steger examined 180 at Nashville, Tenn., all of which were also free. Dr. William Myers examined 330 hogs at San Antonio, Tex., finding trichinæ in but 2, or 0.6 per cent.§

Dr. H. J. Detmers has examined from August to December, 1883, for the Department of Agriculture 3,331 at Chicago; of which 80, or 2.4 per cent., were found to be infected. Of this number 1,126 were from unknown districts of the West; 46, or 4.08 per cent., containing trichinæ; 50 were from Michigan, among which 4 contained the parasite; 831 were from Iowa, of which 19, or 2.27 per cent., were infected; 50 were from Dakota, of which 1 was infected; 520 were from Illinois, among which were 7, or 1.35 per cent., containing trichinæ; 304 were from Wisconsin, with but 2 infected, or 0.66 per cent.; 350 came from Nebraska, having but 1 infected, or 0.28 per cent.; 100 were from Minnesota, and were free from infection.

In the laboratory of the Department of Agriculture specimens from 300 hogs have recently been examined, and of these 5, or 1.66 per cent., were found to contain this parasite.

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* Vieteljahrschrift f. Ger. Med. &c., N. F., XXXVII, p. 345-351.

† A Report on Trichiniasis as observed in Dearborn Co., Indiana, in 1874. By George Sutton, M. D., Aurora, Ind.

‡ Report of American Health Assoc., vol. 7, p. 136.

§ Report of American Health Assoc., vol. 7, p. 136-145.

We have above the records of the examination of 18,889 hogs from various parts of the United States, of which 517, or 2.7 per cent., contained trichinæ. It is evident from these records that a considerable proportion of the hogs from some sections of the country are trichinous, while those from other sections are practically free from infection. While it may be difficult to outline the trichinæ districts and to trace the infected animals to the farms on which they were raised, it is believed that such a study would do much to clear up the origin of this infliction.

In addition to the inspections detailed above, Drs. Osler and Clement examined at Montreal 1,000 hogs from Western Canada, finding 4 infected.* The French inspectors report the examination of 103,528 pieces of American meat, containing 2,080, or about 2 per cent., infected.† The German inspectors, during the year 1880, examined 78,880 pieces of American pork, of which we have record, and found 1,265, or 1.6 per cent., to contain trichnæ; and in 1881 they examined 96,485 pieces, finding trichinæ in 2,414, or 2.5 per cent.‡

Taking all the examinations of American pork thus far made, both at home and abroad, and we have a total of 298,782, during which trichinæ were found 6,280 times, being 2.1 per cent., or 1 to 48.

It would seem that this number of pieces, considering the close agreement between the results reached by American microscopists over this limited territory and those obtained by the inspectors of American pork in Europe, perhaps represents the condition of American pork so far as examined.

In Europe there are some localities where the inspections have shown a greater proportion of infection than the average in the United States. At Stockholm 2,000 hogs contained 58 infected ones, or 2.9 per cent.; at Tannefors 300 hogs contained 10 infected ones, or 3.3 per cent.; and in 112 Bavarian hams 3 were trichinous,§ while Dr. Rine, of Linten, reported 4 infected animals in 45, or nearly 9 per cent.‖ In Prussia, where the only really effective inspection is made, the proportion found infected with trichinæ was, in 1876, 1 to 2,000; in 1877, 1 to 2,800; in 1878, 1 to 2,000; in 1879, 1 to 1,632; in 1880, 1 to 1,460; in 1881, 1 to 1,839; in 1882, 1 to 2,056.¶ The number of inspectors in 1882 is placed at 20,140. Several inspectors at Erfurt were removed on account of incapacity, and complaints were made as to the condition of the microscopes. Indeed, it seems that the village barber is usually trusted with the inspection of hogs in the smaller towns and villages, and that he is required to make but three preparations from each animal.

Frequently, or generally, the specimens for examination are taken from the hams and hard muscles, where the trichinæ are least abundant, instead of from the pillars of the diaphragm and tenderloin, where they are most easily found. Since the great epidemic of trichiniasis in Saxony it seems to be admitted that these inspections are totally inadequate, and in order to make them more efficient the magistrates have awarded sums varying from 15 to 30 marks for each trichinous animal discovered. As a result of the rewards, and possibly of the fear excited by the recent terrible outbreaks of the disease in people, an increased number of infected hogs seem to have been discovered.* These facts, as well as the extraordinary number of people recently infected from eating pork which had been inspected, are sufficient to demonstrate that large numbers of trichinous hogs pass the inspectors without being discovered, and that consequently the figures given above are not a correct representation of the proportion of hogs which are infested with this parasite.

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* An Investigation into the Parasite in the Pork Supply of Montreal, 1883, page 6.

† Chatin, La Trichine et la Trichinose, Paris, 1883, page 217.

‡ H. Eulenberg. Ueber die im Jahre, 1881, auf Trichinen und Finnen untersuchten Schweine. Vierteljahrschrift, f. Ger. Med., &c., 1882.

§ Warfwinge, Nord. Med. Ark., 1875, VII, 3, No. 18.

‖ Meissner, Schmidt's Jahrbücher, No. 130, page 118. Quoted by Glazier Rep., page 68.

¶ H. Eulenberg, Vierteljahrschrift f. Ger. Med., 1877 to 1883.

In nearly every country of Europe hogs have been examined and a certain number found to contain trichinæ, but the records do not seem to have been carefully kept; the data are not fully given, and there is reason to doubt the accuracy of the work. It is difficult, therefore, to reach any satisfactory conclusion as to the proportion of infected hogs. We may safely assert, however, that no country can with reason claim that its hogs are free from trichinæ, while the probability is that European hogs generally are infected in a much larger proportion than is at present admitted.

In some parts of Europe rats seem to have been examined more carefully than pigs; thus in Saxony one-half of the rats from flayers contain trichinæ, and 20 per cent. of all those caught are similarly infected; in Moravia sixteen out of one lot of twenty rats were infected, nine of a second lot of twelve were infected, seven of a third lot of eight were infected. In Klederling, a suburb of Vienna, seven out of forty-seven, and at Untermeidling two out of thirty-one were infected.† In France, where the authorities now deny the existence of trichinæ except as imported, and where one of the reasons for prohibiting American pork is the alleged fear of scattering this parasite over the country,‡ the only outbreak of trichiniasis on record was caused by the flesh of a native hog; and the rats from the ditches and sewers of Paris, examined by Drs. Goujon and Legros, were infected in very large proportion; one lot of thirty-two contained three with trichinæ, and of seventy-two rats, five were full of these parasites.§

EFFECT OF THE CURING PROCESS ON THE TRICHINÆ.

If we admit that about 2 per cent. of American hogs contain trichinæ, it becomes a matter of the greatest importance for us to inquire into the condition of the parasite after it has been subjected to the action of salt a sufficient time to enable the pork to be carried from the packing-houses in this country to the consumers abroad. And here the effect on the consumers is entitled to more weight as a matter of evidence than those scientific experiments which are simply designed to prove the life of the parasite; for the trichina may sometimes still be living but not have sufficient vitality to develop and reproduce itself. Such trichinæ would be perfectly harmless even though the pork were eaten without previous cooking.

In France it is said in the report of Academy of Medicine of Paris, that 95,000,000 kilograms or 200,000,000 pounds of American pork products had been consumed from 1876 to 1881 without causing a single case of disease. And notwithstanding the fact that large quantities of such pork have been consumed for a number of years, the one outbreak of trichiniasis at Crépy, which was clearly traced to a French hog, is the only instance of the appearance of this disease among people that is recorded in that country.

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* Dispatches of A. A. Sargent, American minister at Berlin, to State Department, dated October 26 and November 12.

† Dr. Glazier, Report on Trichinæ and Trichiniasis, Washington, 1881.

‡ J. Chatin, Trichine et Trichinose, p. 153, foot-note.

§ Thèse de Paris, 1866, and Davaine, Traité des Entozoaires, p. 755.

In Germany, where it is the habit of the people to eat pork without cooking, trichiniasis among people is common, and it has been very frequently asserted in some quarters that many of these cases were due to American pork. During the recent terrible epidemic at Emersleben and neighboring towns, Dr. Brouardel, of the Paris Academy of Medicine, went to Prussia to investigate the origin and nature of the disease and learn what he could in regard to the healthfulness of American pork. Not only did he find that these particular cases of trichiniasis were due to German hogs, but such eminent and well-known authorities as Professors Virchow and Hertwig, who have charge of the pork inspection at Berlin, asserted most positively that no case of trichiniasis in Germany had ever been clearly traced to American pork, although the people, as is their habit, persist in eating it raw.*

The so-called outbreak of trichiniasis on board the English reformatory school-ship Cornwall has been much quoted as illustrating the danger of American salted pork, but when closely investigated it proves to be an illustration of jumping at conclusions without evidence, as always seems to have been the case where trichiniasis has been attributed to our meats. This outbreak of disease occurred between September 23 and October 23, 1879, and forty-three boys were attacked out of a total of two hundred and sixty-two boys and fifteen officers on the ship. The idea that the disease was trichiniasis seems to have been an afterthought, for the only examination made was of the body of one of the boys two months after it had been buried. Doctors Powell and Cory thought they found trichinæ in the muscles, and concluded the disease must have originated from the American pork, which was used on board the ship; but no examination of this pork appears to have been made, and we are not at all certain that English pork was not used as well. Fortunately, specimens taken from the corpse were submitted for examination to that well-known scientist, Dr. Charlton Bastian, and he pronounced the worms not trichinæ at all, but a hitherto unknown nematoid, which he classed with the genus *Pelodera*, calling the species *Pelodera setigera*. Dr. Cobbold, who is one of the very best authorities on this subject, asserts very positively that the worm was the *Pelodera teres*, and had probably invaded the body after death.† As this worm has never been known to exist as a parasite in the hog, the assumption that the disease was produced by eating pork is an entirely gratuitous one, and the further assumption that it was due to the American pork is evidently without the least foundation.‡

England has been one of the largest consumers of American bacon, hams, and pork, taking even in 1880 and 1881, when this trade reached its largest proportions abroad, five times as much as either France or Germany. Belgium has also been a large consumer. The *Comité consultatif d'hygiène publique de France* said in a recent official report that in order to determine the danger from the use of American pork they had recently made new inquiries in England and Belgium. In England they were told that trichiniasis was so completely unknown that it was never mentioned, either in the newspapers, the hospitals, or in teaching medicine. And this was also the case in Belgium.§

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* P. Brouardel, L'Epidémie de trichinóse d'Ermesleben. Bul. de l'Acad. de Méd., Paris, 1883, p. 1501.

† Veterinarian, 1884, p. 4.

‡ Power, W. H. Outbreak of Fever proved to be Trichiniasis on board Reformatory School-ship Cornwall. Rep. Med. Off. Local Gov. Bd., 1879. London, 1880.

§ H. Bouley, Bul. de l'Acad. de méd., Paris, 1884, p. 33.

We may conclude, therefore, that notwithstanding the enormous quantity of American pork which has been consumed in Europe, there is no reliable evidence that any cases of trichiniasis have ever originated from its use.

We can now consider more intelligently the conflicting testimony in regard to the condition of the trichinæ in American salted meats when they reach Europe. In 1879 it was stated in the German reports that although a very considerable number of examinations had been made at Minden, no living trichinæ had been demonstrated in preparations of American pork.[*] This statement was repeated in 1880 by the same authority.[†] In France, Colin and most others who have experimented with the trichinæ of American meats have found them dead and incapable of producing any injurious effects when fed to other animals. It was also found that even slight salting killed all the trichinæ within two months. Colin concludes, therefore, that the danger from eating American pork, considering the time that it must have been in salt before it can reach Europe, is slight or inappreciable.[‡] Fourment[§] and Chatin[||] have contested these results, but their opinions are so extremely radical as to lose much of their force on this account. It is not impossible that in certain very rare cases the capsules containing the trichinæ may have become so dense or so impregnated with lime salts as to protect the parasites for a longer time than usual against the action of the brine; but the complete innocuousness of our pork as demonstrated by its use on so large a scale in England, France, Belgium, and Germany, with no cases of disease clearly traced to it, is the strongest possible evidence of the destruction of the trichinæ during the process of curing.

Dr. Brouardel, of the Paris Academy of Medicine, who investigated the recent outbreaks in Ermsleben, has furnished new and very important evidence on this point.[¶] He learned that the meat of the diseased hog was chopped and mixed with sufficient salt to preserve it, and those who ate of this meat soonest after the killing of the animal were not only more severely affected, but their symptoms appeared in a shorter time. The animal was killed the 12th of September, and of those who partook of this meat on the 13th 33 per cent. died, while of those who did not eat of it until the 18th and 19th none died. In fact, there was a very regular graduation in the intensity and fatality of the cases when they were classified according to the number of days which had elapsed between the killing of the animal and the eating of the meat. It was very evident that the parasites were rapidly losing their vitality and their power to produce disease. M. Colin thought this was due to the effects of the slight salting, and M. Brouardel seemed willing to admit this.

If, then, so marked a result is produced in a single week by the slight salting which this chopped meat received, it is very plain that the high degree of salting to which our packed meats are subjected must be sufficient in the vast majority of cases to completely destroy all trichinæ and to make the meats perfectly safe. The question cannot be narrowed down for this reason to a comparison of the proportion of animals affected with trichinæ in America and Europe, even if this were actually determined, which is not the case, but it necessarily turns on the healthfulness of the meats of these countries at the time when they are offered for consumption. And when the matter is viewed from this stand-point the very great superiority of American salted meats over even the inspected German hogs is too apparent to be questioned by unbiased scientific men.

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[*] Eulenberg, Vrtljhrscht. f. ger. méd., 1879.

[†] Loc. cit., 1880.

[‡] G. Colin, Comptes Rendus, xcvi (1882), 886–'8.

[§] L. Fourment, Comptes Rendus, xciv (1882), 1211–'13.

[||] J. Chatin, La Trichine et la Trichinose, Paris, 1883, 164–190.

[¶] Brouardel, Bul. de l'Acad. de méd., 1883, 1501.

In the latest discussion on this subject in the Paris Academy of Medicine M. Proust said:

The question is not a determination if American meats contain trichinæ, but in what condition these trichinæ are found; if they are alive or dead; if they are injurious or not; in a word, if the consumption of American salted meats is dangerous or not to the public health.

In this connection I ask permission of the academy to read a passage from a most interesting letter that I received this morning from Dr. Gibert, a health officer and distinguished sanitarian of Havre:

"In 1881," says M. Gibert, "American salted meats entered largely into the food supply of the working class of Havre; but in regard to this it is important to divide the consumers into two classes:

"1. The people buying American salted meat for family consumption always cooking it and never eating it raw. The inhabitants of the quarters of Eure and Saint François nourished themselves exclusively with it.

"2. The workmen employed in handling the packages of salted meat, who during fifteen years continued to eat this meat raw. MM. Bouley and Chatin could easily have seen in their walks on the wharves of Havre workmen breakfasting on a piece of bread and a slice of raw bacon, eating not only the fat but all parts of the meat. Any one could repeatedly see these workmen day after day, even when at work, eating pieces of raw salt pork without fear.

"It is certain, then, that at Havre, for more than fifteen years, hundreds of workmen consumed salted meats trichinous as well as not trichinous, and never during this long period of years has a single workman been incommoded by this food.

"No physician in Havre has seen a disease resembling in the least the Ermsleben disease described by MM. Brouardel and Grancher. Such an assertion demands some proofs which it is easy for me to give.

"I would remark, in the first place, that if the meat consumed raw had affected the health of the workmen employed in such large numbers handling salted meats the directors would have soon discovered the vacancies in the ranks of the workers. After an investigation carefully made by me, and which it is easy to make anew officially, it was found that never had there been a knowledge of such a fact in any of the large importing houses of Havre.

"The workmen, after the arrival of the Paris savants, took pleasure in eating the pieces themselves that were pronounced trichinous, so certain were they of their perfect harmlessness. And neither during the stay of these gentlemen at Havre nor afterwards was there a single case of disease, or even a simple indisposition.

"In the second place, I would remark that during this period of fifteen years we have had no serious epidemic of typhoid fever. That of 1880–1881, the only one at all serious which has occurred in our city, affected the quarters occupied by the well-to-do or rich people, while the quarters where the American meat was consumed suffered very little. There was then no possibility of an error on the part of the physicians, even if such a gross error could have been committed.

"It follows, from the facts that I have just related, that the salting of American pork is sufficient to kill the trichinæ; and if, in addition to this cause of security which has been experimented upon by the workmen of Havre for fifteen years, we add the cooking, as it is practiced everywhere in France, the conclusion is forced upon every one not prejudiced in advance that American salted meats are absolutely incapble of producing trichiniasis in the consumers."

In the same discussion M. Leblanc said:

The discussion appears to me exhausted; however, I ask permission of the academy to communicate in support of the note of Dr. Gibert the following observation. It was furnished to me by the principal meat inspector of Paris:

The veterinarians under his direction examined during six months in 1881 5,000 kilograms (11,000 pounds) of American salted meat per day. Sixty thousand kilograms were seized as trichinous, and a large part was shipped to England. During these six months the employés and draymen of the dealers in salted meats who came to the station of Batignolles ate meat in presence of the inspectors which was notoriously infected with trichinæ. Not one of them became sick.

One of these, M. R., employed by Calman, 11 Rue Bergère, was accustomed to this, and took pleasure in eating the parts of the pork in which the microscope had demonstrated the presence of trichinæ. To-day, after three years, he is well; more than this, having been received at Beanjon as a patient of our colleague M. Tillaux, for a fractured arm, and having related his bravado, he was the subject of a special examination; his muscular tissue was recognized to be healthy, and no trichinæ could be found in it.*

EFFECT OF COOKING ON TRICHINÆ.

If the trichinæ of American pork are destroyed by the curing process in a time much shorter than is necessary for such meats to be shipped from our packers to any of the consumers in Europe, it may seem superfluous to go into a consideration of the effect of cooking, and yet it is not wholly so. Under certain conditions it would appear that the trichinæ do resist the curing process for a considerable time; and though these conditions occur so seldom that uninspected salted meats are much safer than inspected fresh ones, it is still worth our while to inquire if perfect safety against infection cannot be guaranteed where a reasonable degree of cooking is practiced.

There is some conflict of opinion as to the temperature necessary to destroy trichinæ, and yet the results of experiments do not differ so widely. Vallin† concluded that a temperature of 54° to 56° C. (129 to 133° F.) kills most of them, and that 60° C. (140° F.) is safe. Fiedler's experiments show that trichinæ are quickly killed at 62.5° C. (144.5° F.) Fjord's investigations show that the interior of a ham weighing 8 pounds reaches 65° C. (149° F.) after boiling two hours and seventeen minutes; one weighing 10 pounds, after three hours and six minutes; one weighing 14¾ pounds, after four hours and eleven minutes; and one weighing 16 pounds after four hours and thirty-seven minutes. Vallin found that a ham weighing 12 pounds had an interior temperature of 65° C. after three and a half hours' boiling. These results, therefore, correspond very closely.‡ Hein found that a 2.2 pound roast reached a temperature in its interior of 69° C. after one and a half hours. Rupprecht observed that rapidly fried sausage only had an interior temperature of 53.5° C., and was still capable of producing infection. Colin§ had a steak weighing half a pound boiled for ten minutes, when its appearance on cutting was white, without any red points. It still contained living trichinæ, however, which, being fed to a bird, were afterwards found developed in the intestine.

The indications from these experiments are that while fresh meats may not always be cooked sufficiently to kill trichinæ, salted meats are almost invariably cooked for more than the necessary time. This conclusion seems also to be borne out by the experience of people in all parts of the world. Trichiniasis from cooked meats is an exceedingly rare disease. In the United States, where pork in its various forms is consumed to as large an extent as in any part of the world, and where more than one-fourth of the hog product of the world is eaten, it is seldom, indeed, that we hear of any infection among our native population, because the habit of eating raw meats, particularly when fresh, does not prevail. The few cases which occur from time to time are nearly always among Germans and are traced to the ingestion of pork in some form, which has not been cooked at all.

In this connection Dr. Brouardel brought out a very interesting fact in his investigation of the epidemic at Ermsleben. No cases of disease occurred there except with those who ate the meat raw. The family of

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* Bulletin de l'Académié de Médecine, 1884, No. 6 (February 5), pages 241 and 247.

† E. Vallin. De la résistance des trichines à la chaleur et de la température centrale des viandes préparées. Rev. d'hyg., Paris, 1881, III, 177-182.

‡ C. Hein. Rep. of a case of trichiniasis, with remarks on diagnosis and prophylaxis. Mitth. d. Ver. d. Aerzte in Nied Pest, 1883.

§ G. Colin. Sur les trichines. Bulletin de l'Acad. de méd., 1881, 243.

Herr Heine, the mayor of Ermsleben, consisting of five persons, consumed some of this same meat in the form of sausage on the 15th of September. The sausage was cut in pieces about 1½ inches in diameter and was cooked by placing in boiling water for only five minutes. Not one of this family suffered in the least degree, but the cook who ate a small piece of the sausage before it was cooked contracted the disease. Boiling for so short a time has never heretofore been considered sufficient to destroy this parasite, and yet in this instance it undoubtedly protected the consumers from the infection.

At the session of the Paris Academy of Medicine, January 29, 1884, a report was presented by the special committee appointed to consider (1) the report of M. Brouardel in regard to his mission to Ermsleben, (2) the communication of M. Grancher on the symptoms and pathological anatomy of the epidemic of Ermsleben, and (3) a letter from the minister of commerce asking the advice of the academy in regard to the question of trichiniasis. In this report appear the following points of interest:

> A considerable invasion of German rats has been noticed of late years after the freezing of the Rhine, and M. Brouardel no longer sees the same species of rats at the morgue that he formerly found there. M. Laboulbene and M. Colin had occasion to observe quite a large number of trichinous rats that had been found in the sewers of Paris. Finally, we are also invaded by the trichinæ which penetrate France with the German hogs, which are infected in the proportion of 1 to 1,000 to 1 to 2,000.
>
> Without doubt we do not know precisely, scientifically, the degree of activity of the trichinæ according to the time that they have been encysted in American meats, their vigor, the condition of the meat and the stage of the curing—desiderata which future experiments can alone supply.
>
> The same ignorance exists in regard to our own hogs—are any of them trichinous? If so, what proportion? What is the condition of those in the suburban zone surrounding Paris which, nourished like the sewer rats, may be trichinous like them?
>
> It remains for us to speak of the action of American salted meats in the production of the epidemics of trichiniasis in Germany which have been observed at Dusseldorf, Rostock, and Bremen, but our information in this respect is entirely insufficient, and according to the indications that M. Brouardel has already presented to the academy the exotic origin of these epidemics is denied by several of the principal German savans, and particularly by M. Virchow. Our Government might address the German Empire in regard to this in order to have official documents; but the results of such an inquiry have for us, from the standpoint now under discussion, an almost secondary importance. If in fact we, like the English and the Belgians, have been free from epidemics of trichiniasis although we received salted trichinous meats and even fresh or slightly salted pork of German origin more dangerous than that which comes from Chicago or Cincinnati, since it had only to cross the Rhine and the Vosges, this is because our culinary habits are entirely different. Let us remark also that in America it is above all the Germans who are affected with trichiniasis—an additional proof of the effect of culinary habits.
>
> But already we can conclude from the developments which precede that no case of trichiniasis having been noticed either in France or in England from the consumption of American salted pork, the importation of these meats may be authorized in France. This decision has been reached by the commission by a vote of 5 to 1.

At the same session of the academy M. Lunier presented the following table, showing the importation of salt-pork products for eight years, in kilograms:

Year.	United States.	England.	Germany.	Other countries.	Total.
1876	3,269,960	1,875,736	702,530	1,047,920	7,896,146
1877	12,462,078	1,889,905	994,334	1,078,505	16,424,722
1878	28,102,290	1,454,884	921,167	1,314,437	31,792,778
1879	31,784,913	1,107,691	1,039,673	1,742,854	35,675,131
1880	34,246,195	1,233,228	846,174	2,387,671	38,713,268
1881	17,123,767	686,525	723,465	1,182,474	19,716,231
1882	4,611	1,109,870	1,055,783	1,074,259	3,244,523
1883	52,396	....	1,136,793	2,091,779	8,274,966

It follows from this table that the importation of American salted meats, which had augmented rapidly from 1876 to 1880, began to diminish in 1881, after the decree of February 18, 1881, which prohibited the importation of salted meats from the United States into all the French territory. The 4,611 kilograms which appear in the year 1882 came from a stock taken from the warehouses December 31, 1882. The 52,396 kilograms of 1883 were introduced after the promulgation of the decree of November 27, 1883, which removed the interdiction.

The same gentleman presented the following table, which shows the number of live hogs imported into France in the years 1877 and 1882, and the countries from which they came:

Countries.	1877.	1882.
Germany	15,983	16,165
Belgium	57,806	68,716
Spain	4,234	2,204
Italy	66,366	9,567
Switzerland	964	1,586
Other countries	941	913
Total	146,294	99,148

In this connection he remarked, "It is then, to-day, Belgium and Germany which furnish us the greatest number of living hogs. Is it not from this direction that there is reason to fear the invasion of trichinæ and trichiniasis?"*

TRICHINIASIS IN AMERICA AND EUROPE.

The number of cases of trichiniasis occurring among people in the United States is actually very small. The records of these have not been brought together in a thorough manner, but, as far as we have been able to ascertain, the disease has never occurred in more than three or four localities in a single year, and during the last twenty years there does not appear to have been more than thirty different outbreaks. In none of these outbreaks have a sufficient number of people been attacked to allow of the term epidemic being applied to them in any proper sense of the word. Usually but two to four people have been affected at a time, and never, so far as we have been able to learn, more than ten. All have resulted from eating raw or very imperfectly cooked meat, and in very few of the instances had the pork undergone any preliminary curing. Some of the cases reported as trichiniasis were never demonstrated to be this disease, but seem to have been the result of poisoning by meat which had been preserved without sufficient salting until it had undergone partial decomposition. It has long been known that extremely virulent poisons are produced during putrefaction of flesh, and the effects of these have been observed so often in Germany when sausages were eaten that they have received the special name of *wurstgift* or sausage poison. This does not seem to be understood by many American physicians, and so nearly every case of sickness arising from the consumption of the raw or imperfectly cooked flesh of hogs in the various forms in which it is preserved is reported as trichiniasis. So that while it may be true that some cases have not been reported or brought to our notice it is equally true that not all the cases reported as trichiniasis were really due to trichinæ.

In the debate which occurred in the French Senate June 20, 1882, M. Festelin referred to seven epidemics produced by American pork.† One of these so-called *epidemics* consisted of a single case of the disease which occurred in New York. Another was the disease on the English ship Cornwall, which was at first supposed to be typhoid fever, and was only decided to be trichiniasis when a body was exhumed two months after burial, and worms, which the examining physician took to be trichinæ, were found in the muscles. But we have it on the very best authority that these worms were not trichinæ, and there is no evidence even that they caused the disease. The most probable theory is that they gained access to the body after burial. No trichinæ or other parasites were found in the American meat consumed on this vessel.

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* Bulletin de l'Académie de Médecine, 1884, pp. 189–211.

† Chatin, La Trichine, &c., p. 210.

Another epidemic he referred to as having occurred at Bremen, in which forty persons became diseased from eating an American ham. M. Chatin has mentioned this outbreak again and again, and insists that it is a demonstration of the dangerous character of American meats. The charge was so serious that it has been investigated as carefully as possible. M. Testelin does not give the authority who is responsible for this statement, nor does he so much as say in what year the outbreak occurred. M. Chatin is more definite, however, and says the disease was observed in 1875.* He refers to the *Traite d'hygiéne publique et privée*, by Proust, published in 1877, as his authority for asserting that forty persons were affected at this place as the result of eating an American ham. By consulting the yearly health report of Bremen for 1875 we find that no cases of trichiniasis in man are recorded during that year. There is simply a statement that two trichinous hogs (native animals) were discovered near Bremen. The outbreak of trichiniasis referred to by Testelin and Chatin seems to have been one that occurred at Hastedt, near Bremen in 1874, during which forty-two persons suffered, but all recovered. This epidemic was first announced August 15, and was caused by eating the flesh of a hog that had been slaughtered July 31. The diagnosis was confirmed by microscopic examination of a piece of muscle from one of the sufferers.† There had been a habit here, as in most other parts of Germany, of attributing all cases of this disease to American pork without investigation, and this may have been the origin of the story so industriously circulated by M. Chatin. At all events, this is the only extensive epidemic of trichiniasis which is recorded as occurring at or in the vicinity of Bremen from 1873 to 1877, inclusive, and there is no question but that this was caused by a native animal.

One of the other epidemics referred to occurred in Madrid, and another in Liege, in Belgium. It is doubtful if in either case the trouble was traced to our pork; but no facts in regard to them have yet been obtained.

M. Chatin, in his recent work on trichinæ and trichiniasis, states that "such is actually the frequency of trichiniasis in the United States that the newspapers consider themselves happy if they have but a few deaths to record each week." Whether any one in France believes this remarkable exaggeration may be considered questionable, but still it is seriously advanced by a scientific man as a reason for prohibiting our pork. Those who see the American papers know very well that not only weeks but months elapse when no deaths are recorded from this cause. And if it were not for our foreign population, who have brought with them their dangerous habits of eating uncooked pork, America would be as free from trichiniasis among her people to-day as is France.

When we examine the records of Germany, however, we find that, in spite of the small proportion of infected hogs which they admit, in spite of the inspection, there occur a very large number of cases of this disease. In 1877 there were 16 in Königsberg, 1 in Potsdam, 6 in Berlin, 98 in Stettin, 1 in Oppeln, 61 in Merseberg, and 52 in Minden. In 1878 there were 27 in Königsberg, 8 in Marienwerder, 102 in Potsdam, 50 in Stettin, and 30 in Merseberg. In 1879 there were 55 cases in Königsberg, 93 in Frankfort-on-the-Oder, 82 in Berlin, 7 in Marienwerder, 3 in Schleswig, 60 in Erfurt, 7 in Merseberg, and several in at least three other places. In 1880 there were 149 in Merseberg, 83 in Erfurt, 49 in Frankfort-on-the-Oder, 3 in Marienwerder, 29 in Königsberg, and 16 in Berlin. In 1881 there were 3 in Marienwerder, 15 in Berlin, 10 in Frankfort-on-the-Oder, an indefinite number in Posen, 4 in Stettin, 148 in Merseberg, and 58 in Erfurt. In 1882 there were 3 cases in Berlin, 60 in Cologne, 4 in Merseberg, 4 in Heiligenstadt, and several in Posen.* In 1883 the remarkable epidemic in Saxony occurred from eating pork which had been slaughtered and inspected in the town of Ermsleben. In Ermsleben 257 persons contracted the disease, and 50 died.† In Deesdorf there were 40 cases and 9 or 10 deaths; in Nienhagen 80 cases and 1 death. There were a number of other epidemics during the year, the statistics of which have not yet been published, but we have already recounted sufficient to show that trichiniasis is incomparably more frequent in Germany than in America.

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*Chatin, La Trichine, &c., p. 165.

†Dritter Jahresbericht über den öff. Gesundheitszustand, &c., in Bremen, in Jahre 1874.

Indeed, for the seven years from 1877 to 1883, inclusive, the very incomplete statistics given above show that 1,835 people contracted the disease, being an average of 262 cases per annum. When in addition to this we consider that the United States is really the greatest pork-eating nation in the world, that we consume more than four times as many hogs as are raised in Prussia, that a considerable portion of our population consists of Germans who retain their habit of eating raw pork, the wonder is not that we have a half dozen or a dozen cases of trichiniasis in a year, but that we do not have many times this number; and we see no way of explaining the comparative immunity which our people enjoy except by the conclusion that our pork, even when fresh, is not so much more dangerous than the German article as the results of microscopic examination thus far published would lead one to suppose.

REPORTS OF BOARDS OF HEALTH.

The commissioners prepared and forwarded copies of the following circular letter to the various State and city boards of health:

> With a view to ascertaining the extent to which trichiniasis prevails in the United States, the following questions have been formulated, to be addressed to the secretaries of the various State and city boards of health.
>
> As it is desirable that the report of the commission should be presented to Congress at once, the undersigned would beg as speedy a response as possible:
>
> 1. How many cases of trichiniasis have come to the knowledge of your board? Please give dates as far as practicable.
> 2. In how many cases was there a microscopic identification of the trichinæ in the human subject and in the suspected meat?
> 3. How many of the cases were fatal?

Responses have been received from thirty-nine of these boards; of this number twenty-two give negative replies; they were as follows: Alabama, Arkansas, California, Delaware, District of Columbia, Kentucky, Louisiana, Maryland, Minnesota, Missouri, Albany (N. Y.), Ohio, Pennsylvania, Rhode Island, Tennessee, and Virginia.

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* Eulenberg, Vrtljrscht. f. ger. Med., N. F., XXVIII-XXXVII.

† Deutsche med. Woch., 1884, No. 1, p. 7.

The secretary of the State board of Alabama says that no case has ever occurred in that State, though infected meat has been reported.

The secretary of the health department of Maryland says that after an examination of the records from January 1, 1834, to December 31, 1883, covering a period of fifty years, he finds no cases of trichiniasis reported.

The health officer of Cleveland, Ohio, says that in 1872-'73 there were some cases of trichiniasis reported in that city, but no definite record of them can be found.

CASES OF TRICHINIASIS REPORTED.

Cases of trichiniasis are reported by the following boards, viz:

Connecticut.—Three in 1882, all from the same pork and at the same time. The suspected meat was examined by Mr. C. W. Chamberlain, of Hartford, secretary of the board, and others. No deaths resulted.

Illinois.—The registrar of vital statistics for the city of Chicago says:

> Three deaths from trichiniasis have occurred in this city within the last ten years. Two of these occurred in December, 1880, and the last one in January, 1882. There was a microscopic identification in the two first cases and probably also in the last.

The secretary of the State board of health says:

> Between 1866 and 1881 there had been seven outbreaks of trichiniasis in Illinois, resulting in eleven deaths out of some seventy or eighty cases. Since 1881 there have been two deaths in Chicago; three outbreaks, resulting in three deaths, and some twenty-five or thirty cases in 1883, and one in Fulton County in 1884, details of which have not yet been received. In nine of the fatal cases the disease was identified by microscopic examination of the human subject, and of the suspected meat in all cases.

Iowa.—The Secretary of the State board of health reports as follows:

> Since 1880, in compiling deaths, I have found 3 recorded as from trichiniasis; in 1880, 2 in Adams County, probably genuine cases; in 1881, 1 in Butler County, a very doubtful case. None of the cases were identified by microscopic examinations.

Massachusetts.—The health officer for the State board of health for Massachusetts says:

> The number of cases of trichiniasis that have come to the knowledge of the board are as follows:

	Cases.
In Saxonville, Mass., February, 1870	3
In Lowell, Mass., March, 1870	6
In Framingham, Mass., December, 1872	3
In Becket, Mass., May, 1873	4
In Wakefield, Mass., March, 1880	3
Total number of cases	19

> One death occurred in the outbreak at Saxonville.
>
> Identification of trichinæ was made in the fatal case at Saxonville by *post mortem*. In the Lowell cases trichinæ were identified in the pork, as also in the Framingham cases. In the seven remaining cases at Becket and Wakefield the evidence was symptomatic and by exclusion, *i. e.*, members of families who had not partaken of uncooked pork were exempt from infection. The evidence, however, was satisfactory to the board.

Michigan.—The secretary of the State board of health of Michigan gives the following detailed report of ten outbreaks of trichiniasis in that State since December, 1866:

> 1. A fatal case of trichiniasis occurred in Detroit, Mich., in December, 1866. Upon *post mortem* examination large numbers of trichinæ were found in the abdomen and a lesser number in the muscles of the leg. (Dr. Herman Kiefer.)
>
> 2. Five cases occurred in Port Huron, Mich., in January, 1874, with 2 deaths. The cases occurred in one family, and were caused by eating salted smoked ham. (Reported by Dr. M. Northup, Port Huron, Mich.)
>
> 3. Several cases of trichiniasis occurred near Flint, Mich., in the fall of the year 1875. Cases also occurred there in June, 1876. In the last outbreak, at least, trichinæ were found by microscopic examination in the meat, and also in a particle of muscle

of the leg of one of the patients. (The cases were in the practice of Dr. A. B. Chapin, of Flint, Mich., now of Detroit.)

4. Five cases occurred in Otsego Township, Allegan County, Michigan, in February, 1877. All recovered. All were in one family, the members of which had eaten raw ham. Members of another family ate a small quantity of the meat. and were also sick, but recovered without medical attendance. Trichinæ were found in the meat by microscopic inspection.

5. Three cases, with one death, occurred in the city of Iona, Mich., in 1878, in the family of Mrs. Strunck. The meat was eaten raw. A microscopic examination of the meat was made, and, I believe, trichinæ were also identified in the human subject.

6. I am informed that cases, and one or more deaths, occurred in the vicinity of Iona, Mich., in the summer of 1880, in the practice of Dr. H. B. Barnes. Trichinæ were found in the pork, and also, I believe, in the muscles of those who died.

7. Five cases and two deaths occurred in the family of a German named Rumsock, in Lansing, Mich., in the last of January, 1881, and the first of February, 1881. The cases were in the practice of Dr. Dolan, of Lansing, who has since died. Great numbers of non-capsuled trichinæ were seen by Dr. George E. Ranney, myself, and others in the muscles of a boy who died. (I still have slides showing the trichinæ as taken from the muscles of a person in the family who died.) Part of the meat was eaten raw. It was not examined for trichinæ; but the hog was sick and was killed to avoid loss of the animal.

8. Five cases of trichiniasis occurred in Vickeryville, Montcalm County, Michigan, in December, 1883. All resulted from eating the flesh of one hog. One death occurred. The boy who died had at different times eaten small pieces of pork only partially cooked by holding them on a fork before the fire. He had also eaten raw, on several occasions, little bits of sausage made from the flesh of the hog. Two others had eaten the meat cooked, but probably rare-done. The other person had eaten raw a little bit of sausage, perhaps half an ounce, but the rest of what he ate was well cooked. Trichinæ in abundance were found in the pork, specimens of which are now in this office. (Reported by Dr. J. Tennant, Carson City, Mich.)

9. Four cases occurred in January, 1884, in the city of Niles, Mich. One death has occurred and three persons are now seriously sick. The pork contains great numbers of trichinæ, as determined by observations made at Niles, Ann Arbor, and in this office.

Further details as to the five cases occurring at Port Huron, and also of the case at Detroit, are given in the published report of this board for the year 1875; and of those occurring in Otsego, in the report of this board for the year 1877.

Such answers as I am able to give to the four questions asked by your Commission have been summarized from the foregoing particulars, and are given with each question, as follows:

"Question 1. How many cases of trichiniasis have come to the knowledge of your board? Please give dates as far as practicable.

"Answer 1. I am able, at this time, to send you facts concerning *ten* outbreaks of trichiniasis occurring in Michigan. In three of these the number of cases was not stated. In seven outbreaks there was a total of twenty-eight cases. The dates are given in most instances in the report accompanying this.

"Question 2. In how many cases was there a microscopic identification of the trichinæ in the human subject and in the suspected meat?

"Answer 2. In two outbreaks the observer did not state whether any microscopic examination was made. It is believed that the human muscles were examined in five of the outbreaks, and in each of them trichinæ were found; in three of these it is positively known that trichinæ were found present. In seven outbreaks the pork was examined, and in each of the seven trichinæ were found in the pork. In the other outbreak. microscopic examination was made only of the muscles of the human subject, in which trichinæ were found.

"Question 3. How many of the cases were fatal?

"Answer 3. In one outbreak it was stated that there were no fatal cases; in one outbreak, where fatal cases occurred, the number was not stated; in one outbreak it was not stated whether any fatal cases occurred; in another outbreak, now in progress, one death has already occurred; and in six other outbreaks seven deaths were reported; making in all, in which the number was reported, eight deaths."

New Jersey.—The records of the State board of New Jersey extend from January 1, 1875, to date, a period of about nine years. During this period but one death from trichiniasis has been reported. This was in May, 1881, and was a female. The method by which the diagnosis was determined is not known.

New York.—The following report is furnished by the secretary of the board of health of New York City:

A few cases of alleged trichiniasis have come to the knowledge of the board, but there is no record of the number or dates.

In two families, during the past eighteen years, there has been, within the knowledge of this board, microscopic identification of trichinæ in the human subject.

During the past ten years five deaths attributed by attending physicians to trichiniasis have been reported to this board: one in 1875, one in 1878, one in 1880, and two in 1881.

The secretary of the board of health of Brooklyn, N. Y., says that five cases of trichiniasis have occurred in that city, of which a full history may be found in the proceedings of the Medical Society of Kings County for 1879.

North Carolina.—The secretary of the State board says that an equivocal case of trichiniasis occurred in Wilmington, in that State, but no microscopical examination was made. It was in the person of a boy of German parentage, habituated to using raw ham.

West Virginia.—The health officer of the city of Wheeling reports five cases—father, mother, and child of same, aged two and one-half years, and two females of other families. All ate of same meat—raw smoked ham—on March 4, 1870. No microscopic identification was made in the human subject.

Wisconsin.—The secretary of the Wisconsin State board of health says that some cases of trichiniasis have occurred in Milwaukee, but does not give the number or dates.

CAUSE AND PREVENTION OF TRICHINÆ IN SWINE.

The worm known as the *Trichina spiralis* has no stage of its existence outside of the animal body, and cannot multiply or even remain alive for any considerable time, so far as has ever been ascertained, after it quits its host. Every infected animal must become infected either by eating the muscular tissue of another animal which has previously obtained the parasite in the same way, or possibly by taking food which has been soiled by the excrement of an animal recently infected. It is generally admitted that eating flesh which contains the parasite is the most frequent, if not substantially the only, way in which trichinæ find their way into the body. A few cases have been advanced to show that pastures and feeding places may be infected and be dangerous for a considerable time; but, according to our present knowledge of the natural history of this parasite, it may be doubted if many instances of this kind occur.

When meat containing trichinæ is taken into the stomach the capsule or cyst which surrounds the worm is dissolved by the digestive liquids, the parasite is set free, develops into its mature form, the females are impregnated, and each gives birth to one thousand or more young. The young trichinæ penetrate the intestinal walls and find their way into the various muscles of the body, while the mature worms, and doubtless many of the young as well, are voided with the excrement. Now, it may be admitted that a large number of the mature parasites will be passed from the bowels before they have brought forth their young, and that if taken into the stomach of another animal the act of reproduction would continue; but it seems doubtful if enough of such mature worms would be consumed in this manner to cause any serious infections. We do not know, however, how long the worms are able to live outside of the body in this developed condition; if they can exist but a few days the danger from them would be very slight, but if this period can be prolonged for weeks or months the danger would be more serious, and we might have at least a partial explanation of the many cases of infection occurring where the condition of life among the hogs seems to be all that could be desired.

The young trichinæ or larvæ which are produced in such enormous numbers in the intestines within a few days after infected meat has been eaten, and many of which are doubtless voided with the excrement, are practically incapable of dangerously infecting grounds or feeding places. These cannot reproduce themselves until they have found their way into the muscular system, and have been encysted for a time, so that even a considerable number of such larvæ taken into the stomach would produce no appreciable effects.

In the present condition of knowledge the tendency is to conclude that by far the larger part of trichinous hogs are infected by eating the flesh of some animal which has previously been infected in the same way. Trichinæ cannot develop or live for any considerable time in the bodies of insects, cold-blooded animals, or birds, and, consequently, the infection must result from some of the warm-blooded animals, which either habitually or occasionally eat flesh. Among these cats, rats, and mice are the ones most frequently suspected; but an inquiry into the conditions under which hogs are raised in the West has led us to doubt if the infection could occur in any considerable number of cases in this way. Hogs are usually kept in grass fields, where rats and mice are not common, and where cats certainly do not abound, and in no part of the hog-raising country is it a custom, so far as could be ascertained, to run the hogs in corn-fields, where there would be an opportunity of their finding rats and mice.

It has been charged that there was a custom of feeding the hogs which died from disease to the well animals, and that this accounted for the trichinous infection. After an extensive investigation, however, we feel authorized to state that this assertion is not correct. Such a practice seems to have been followed to some extent a half dozen or more years ago, but as the contagious character of hog cholera became better understood, and as the demand increased for the cheap grease rendered from such dead animals, they were more generally sold to rendering establishments at a price considerably beyond what they would be worth for animal food. The trichinæ of to-day must therefore be acquired from some other source than the hogs which die upon the farms.

The French and German authors have not hesitated to assume that our hogs were infected by feeding upon offal from the slaughter-houses, but this assumption could only have been made in complete ignorance of the actual condition of affairs in the hog-growing sections of the country. Practically all of the hogs which go to the packing-houses are raised upon farms miles, and generally hundreds of miles, from any large city where offal could be obtained, and they are never fed upon anything but vegetable food. The blood and offal at the large packing houses is dried at a high temperature and sold for fertilizers, and is never fed to the hogs even in the stock-yards. The foreign microscopists have the proof of this in their own hands, if they would only give the matter a little consideration before accepting the absurd statements of ignorant and prejudiced parties.

During the killing season as many as 60,000 hogs are received at the Chicago stock-yards in a single day, and it is evident that it would be impossible to furnish accommodations for holding this enormous number for any considerable time before killing. Now, the trichinæ which are found in American pork are in the vast majority of cases encysted, and for this condition to be reached time is required, and much more time than it is possible to hold hogs in the cities where alone offal for feeding them can be obtained. It is four weeks after infection before cysts are formed, and it is six weeks to two months before they reach the condition in which they are generally found by the microscopic examination of our meats. While we know from our own observation in all the cities where hogs are packed that the animals are not fed upon offal previous to killing, we have here in the condition of the trichinæ themselves the best and the most incontrovertible evidence that the animals were not infected by offal fed while they were held at the packing-houses before slaughter.

It is evident from what has just been said that we are unable at present to give a satisfactory explanation of the manner in which Western hogs become infected with trichinæ, for the conditions of life, at least so far as we are able to see, appear in the vast majority of cases to be all that can be desired. The infected hogs must be traced to the counties from which they come, and even to the farms on which they are raised, and the conditions studied as they exist on known infected premises, before it will be possible to give a solution to this difficult question. And until this is done no effectual rules for prevention can be formulated further than in a general way to recommend that the hogs have no access to any animal-matter except what has been thoroughly cooked.

SALT USED IN PACKING.

The salts most generally used in packing in the United States are the Syracuse solar salt, of which 2,500,000 bushels were manufactured last year; Michigan solar salt, of which 50,000 bushels were manufactured last year, and Turk's Island salt. For rubbing hams either the ground solar salt or the Syracuse factory filled dairy salt is employed.

Some years ago the War Department of the United States Government caused some practical tests to be made by having meats packed with Turk's Island and Syracuse solar salt. The meats were afterward sent to the different forts on the sea-coast, on the Gulf of Mexico, and in the interior.

The result of the experiment demonstrated the fact that the Syracuse solar salt is equal in every respect for packing purposes to the imported Turk's Island sea salt, and since that time the United States Government has required that beef and pork packed for the Army and Navy shall be packed with Turk's Island or Syracuse solar salt.

American packers fully appreciate the importance of good salt, and are very careful in securing the best brands.

The following analyses of American and European salts show that the salts used in this country compare very favorably with those of Europe:

SALT USED IN THE UNITED STATES.

Description.	Sodium chloride.	Calcium chloride.	Magnesium chloride.	Sodium sulphate.	Calcium sulphate.	Magnesium sulphate.	Insoluble matter.	Water.
1. Turk's Island sea salt	96.760	....	0.140	0.640	1.560	....	....	0.900
2. Syracuse, N. Y., solar salt	96.004	0.092	0.089	....	1.315	....	....	2.500
3. Saginaw, Mich., solar salt	95.831	0.356	0.140	....	0.316	....	....	3.344
4. Lincoln, Nebr., solar salt	98.130	....	0.080	0.390	0.250	....	....	1.200
5. Kansas, solar salt	93.060	....	0.240	0.350	1.220	0.180	....	4.950
6. Hocking Valley, Ohio, solar salt	97.512	0.234	0.089	....	....	....	....	2.130
7. Petit Anse, La., rock salt	98.882	0.004	0.003	....	0.782	....	....	0.330
8. Syracuse, N. Y., "factory filled dairy"	97.832	....	0.037	0.026	1.263	0.023	0.120	0.700

ANALYSES OF EUROPEAN SALT.

	Sodium chloride.	Magnesium chloride.	Sodium sulphate.	Potassium sulphate.	Calcium sulphate.	Magnesium sulphate.	Insoluble matter.	Water.
9. Lisbon, Portugal, first crop sea salt	97.075	0.777	....	....	1.538	0.565	0.045	....
10. Lisbon, Portugal, second crop sea salt	94.033	2.151	....	....	1.471	2.337	0.008	....
11. Aveiro, Portugal, first crop sea salt	97.251	1.134	....	....	0.645	0.903	0.067	....
12. Aveiro, Portugal, second crop sea salt	98.618	0.181	....	....	0.640	0.165	0.396	......
13. St. Feliœ sea salt	94.072	0.141	....	....	0.373	0.060	0.116	5.673
14. Velicka, Galicia, rock salt	90.230	0.450	....	1.35	0.720	0.610	5.880	0.85
15. Hall, Tyrol, rock salt	91.780	0.090	....	1.35	1.190	1.210	2.490	1.890
16. Schönebeck, Prussia, Saxony	95.400	0.080	....	0.41	0.720	0.470	....	2.920
17. Dürrenberg, Saxony	92.642	0.719	0.307	....	1.632	....	....	4.700
18. Artern, Saxony	94.835	0.616	0.488	....	1.061	....	....	3.000
19. Halle, Saxony	92.773	0.865	..	....	1.296	....	0.466	4.600
20. Erfurth, Saxony	96.941	0.017	0.049	....	2.093	....	......	0.900
21. Louisenthal	96.866	0.060	....	....	0.964	....	0.110	2.000

AUTHORITIES FOR ANALYSES.

Analyses numbered 1 to 8, of salts used in the United States, were made by C. A. Goessmann, Ph. D., professor of chemistry in the Massachusetts Agricultural College, and published in a lecture on salt and its uses in agriculture, delivered before the State department of agriagriculture in Massachusetts.

Analyses 9, 10, 11, and 12, of Portuguese salt, is from the Mechanics' Magazine.

No. 13 is by Von Kripp, and is from *Oesterr. Zeitschrift für Berg- u. Hüttenwesen*, 1867.

Nos 14 and 15 are by Von Stoba, and are from *Die chemische Mittheilungen, Prag*, 1880.

No. 16 is by Heine, and is taken from Knapp's *Lehrbuch der chemischen Technologie*, 3d ed.

Nos. 17, 18, 19, 20, and 21 are by L. Enders, and are from the *Archiv. der Pharmacie*, Vol. 143, p. 20.

EXTRACTS FROM LETTERS OF CORRESPONDENTS.

DISEASES OF CATTLE.

BLACK-LEG.—Mr. S. Woodsum, jr., of Wilkin County, Minnesota, writes as follows concerning black-leg in cattle:

> I desire to give you my experience with the disease known as black-leg in cattle. Several years ago I lost quite a number of these animals, mostly calves, but I have had a number of cows attacked by the disease. It has invariably been those that were fat that were taken sick. The first indication of sickness is loss of appetite, staring coat and lameness, very sensitive to pressure on the outside of shoulders, and very laborious breathing. A *post-mortem* examination revealed the hind parts normal; the liver very black and tender and breaking into pieces at slight touch; the air passages through the lungs, the heart, and the heart case congested and filled with black blood.
>
> The cause of this disease, I am convinced by experience and observation, is over-feeding. For instance, cows that were milked all winter, and were fed eight quarts per day of corn meal, linseed meal, oat meal, and wheat bran, went through the winter without getting sick; but cows that came to the barn fat and dry, and were not fed anything but hay until after dropping their calf, when they were put on the same feed as those that had been milked all winter, were taken sick in every instance. Again, cows that came to the barn dry and fat, and received a daily ration of feed of the same quality as the other cows, but a much less quantity than before dropping, and then increased in quantity to the same amount as the others, got through without being sick.
>
> My experience has been about the same with calves as it has been with cows. A calf that had been allowed to run with its dam all summer, being very fat, was turned into a field where grain had been harvested and a very luxuriant growth of volunteer grain had sprung up, and it was soon taken sick.
>
> As a preventive we quite often read that salting twice a week will prove beneficial, but this never made any difference with the stock in this vicinity, from the fact that I was about the only one that kept salt where the animals could get it night and morning, and yet I lost as many, if not more, than any of my neighbors. I have always fed more grain than any one else in this neighborhood.
>
> When Dr. Hurn, of the Signal Service, was here, some seven or eight years ago, I described the *post-mortem* appearance of an animal that had died of black-leg. He advised me to try drenching with strong salt brine, and I am happy to say that I accepted his advice, and have not since lost either a cow or a calf. Put one-half pint of salt into a quart bottle, fill with water, shake well, and give about half as the first dose; in about an hour give the remainder, and one hour later repeat. The following day the operation should be again repeated. I have used this remedy in the case of sick horses with satisfactory results.

Mr. L. E. Rowe, of Anderson County, Kansas, recently lost eight head of yearling steers by black-leg. Writing, under date of August 8, he says:

> I have lost eight head of yearling steers out of a herd of one hundred and thirty-five brought from Shannon County, Missouri, to this county this season. The disease is called black-leg, and I can learn of no remedy from local scources. A steer, apparently well yesterday, at evening was observed staggering about, at daylight this morning it was down, at 7 a. m. it was unable to rise, and at 8 a. m. it was dead. On cutting the animal open various dark, bloody spots were found under the skin and involving the tissue. The heart was seriously affected.

An epidemic of black-leg prevailed among cattle in Trego County, Kansas, during the months of November and December last. In the early part of December Mr. Ben. C. Rich, of the above-named county, informed the Department that over one hundred head of cattle had died of the disease in Ogallah Township alone. He had lost eleven animals himself, a neighbor had lost seven, another seven, and still others three or four each. As to the *post-mortem* appearances of the disease, he says:

I was to-day called upon by a neighbor to make an examination of three animals which had just died, of which I send you the following imperfect report:

No. 1. A heifer calf four months old, still fed twice a day on milk and the run of wheat and rye. The first symptoms were weakness in hind limbs and small of back, and a dragging motion of hind legs when attempting to walk. When opened, the blood was found discolored on inside of both hind legs and over the left kidney. The tissue which surrounds the paunch and intestines was also discolored. The small intestines and manifold were very tender and easily torn. There was no bake. The melt was much inflamed and bloody and very tender. Other organs seemed normal. Yellowish froth issued from the mouth.

No. 2. A cow three years old and giving milk. Appeared well yesterday morning when milked. Sickness discovered in afternoon, and died during last night. The first symptom wes lameness in righ hind leg, which was much inflamed at stifle joint and very sore to the touch. On opening, found the whole right horn very much inflamed; flesh very dark and bloody, and over right kidney the same; tissues surrounding the paunch and small intestines much discolored; manifold baked; liver inflamed, and so tender that I could tear it easily with my finger. About a quart of bloody water was found in the cavity of the body. There was no discoloration on front part of body. All other organs seemed normal. The animal was five months gone with calf.

No. 3 A fine, large-grade cow, three years old next spring, and six months gone with calf. This cow did not seem stiff in joints or weak behind previous to death. She ate heartily up to 4 o'clock p. m. yesterday, at which time she was first noticed to be sick. She died during the night. The lesions were confined to the breast and between the front legs. These parts were very much inflamed and the flesh dark and bloody. No discoloration was found on hind parts, back, paunch, or intestines. No bloody water in cavity. The liver seemed all right, but the manifold was baked. The heart was clotted, and the lungs discolored and congested.

Mr. A. E. Hall, of Fort Meade, Dak., writes as follows concerning the ravages of black-leg in that locality:

A disease is prevailing among cattle in this county called black-leg. I examined two animals about twelve hours after death. Upon opening the body the blood seemed almost all gone. What little was left seemed to have settled between the layers of flesh and tissues. The flesh was a little higher colored than beef. The first animal had a swollen shoulder, and when rubbed with the hand emitted a crackling sound. It was full of air-bubbles immediately under the skin. The last stomach was very dry, almost baked. The small intestines leading to the last stomach were green for the distance of three or four inches. The animals show different symptoms. Some became swollen all over, some in one leg, and some not at all. We have lost about a dozen range calves. But yearlings are not exempt from the disease, and sometimes cows are attacked. It does not make any difference as to whether the animals are fat or thin, native or thoroughbred stock. Texas animals seem to be exempt. The disease is very rapid in its work. An animal that is apparently healthy and very lively in the evening may be found dead the next morning. Occasionally one may be stupid for three or four days, but as a rule they die in a few hours.

There is also a disease here among cattle which affects their feet. The foot swells between the toes, laming the animal. In the worst cases the leg swells as high as six inches above the ankle, ulcerates, and exudes a white matter. Sometimes the flesh protrudes from these ulcerated places. It generally disappears of its own accord, but the worst cases either lame for life or kill the animal.

Cattle in Saline, Mitchell, Phillips, Lincoln, Ottawa, and other counties in Kansas suffered severely with this disease during the past season. Mr. Charles E. Faulkner, of Salina, Kans., writing in December last to Hon. J. J. Ingalls, speaks of the disease as being wide spread and disastrous in the counties above named. The following extract is made from Mr. Faulkner's letter, which was kindly forwarded to this Department by Senator Ingalls:

Many thanks for your notice of my communication regarding black-leg among cattle in Kansas. The Department seems to have made no special investigation of this disease. Losses this season will be heavy in the State. Over one hundred head have died in this county (Saline) during the last three months, and the end is not yet. Losses from Mitchell, Phillips, Lincoln, and Ottawa are reported. I hope that Congress will aid the Department in instituting a thorough investigation of the subject, in order that we may become better informed of the nature of the disease, its cause, and preventive measures.

CONTAGIOUS PLEURO-PNEUMONIA IN NEW JERSEY.—Dr. William B. E. Miller, of Camden, N. J., writing to the Department under date of November 10 last, says:

On the 31st ultimo I was summoned to visit a herd of cattle at White House, Readington Township, Hunterdon County, in this State. On the 1st instant I examined some cattle on the farm of Mr. George Clum. Among his herd of about 60 cows I found several infected with pleuro-pneumonia. During the past four months he has lost 22 head. Soon after the first outbreak in the herd he had all the animals inoculated. On another farm owned by Mr. T. W. Pidcock, and tenanted by Mr. Charles Snyder, near Three Bridges, Hunterdon County, is a herd of 65 cows. About the first of March last there was brought from Bradford County, Pennsylvania, to Farmington, N. J., a car-load of cows. From this car-load Messrs. Pidcock and Snyder purchased 13 head. Ten of these animals were put on their farm and three were sent to another farm. The animals then upon the farm were nearly all inoculated. Pleuro-pneumonia broke out among these ten new arrivals, and soon eight had died. One was killed for the purpose of securing virus for inoculation purposes. All the remaining animals were then inoculated a second time. There have been several cases on the farm since, but I was unable to get the exact number at the time of my visitation. Every lot of fresh cattle brought on the place since have immediately been inoculated, but out of every lot one or two animals would either die of the disease or be killed. On another place occupied by Mr. Peter Pidcock, where there had never been a case of contagious pleuro-pneumonia, a bull was brought from the F. N. Pidcock farm that had had the disease but was supposed to have recovered. He infected this herd of 46 cows, and 8 animals died before the contagion abated. All the animals were inoculated as soon as the disease broke out among them. On another farm, occupied by Mr. W. D. Ammerman, 8 or 10 animals died of the disease during the summer months. There are 41 head in one lot and 29 in another on this farm. All have been inoculated. There is one acute case now on this farm. The animal has been inoculated, but still she is suffering from acute pleuro-pneumonia. On still another farm, owned by Mr. Garret C. Gearhart, six animals have been attacked and four have died. All the other animals have been inoculated.

You will observe that every fresh cow brought on to these places, if not soon inoculated, suffered with pleuro-pneumonia, and that while inoculation prevented the animals on the farms from death, the disease has been reproduced in every fresh lot in the genuine form, killing most of those attacked.

CAUSE OF ABORTION IN COWS.—Mr. Robert T. Burbank, proprietor of White Mountain Stock Farm, Shelburne, N. H., writes as follows, under date of March 24 last:

In 1879 fourteen of my herd of thirty-five thoroughbred cows aborted. During the next year I kept strict watch over them, guarding them from storms in summer and keeping them dry and warm in winter, in order that I might discover the cause of the trouble. Notwithstanding my care and watchfulness, five of the animals aborted the following year. I then frequently, with the greatest care, examined my hay fields. In two of these fields had sprung up, from seed I had purchased for high-grade Western timothy, a rank growth of wild rye, and also weeds about two feet high, resembling what is sometimes called "wild strawberry." The seeds of these weeds appeared much like the seeds of timothy. I gathered some of the weeds and sent them to the seed merchant of whom I purchased my hay seed, stating that I was satisfied that I had discovered in this wild rye and its seeds the cause of abortion in my cows, and requesting him to have the seeds examined and report the results to me. He treated the matter with such indifference that I have since ceased to purchase of him.

The hay cut where the wild rye and those weeds grew in the following year (1881) I put in my young-cattle barn, and did not allow my cows to eat any of it. I have since raised nearly all my own hay seed, and last year only one of my cows aborted. This year all have escaped. I already have seventeen beautiful, healthy calves. Their dams are also perfectly healthy and in good condition. Several years since I expressed the opinion that there must be something in the hay similar to ergot that caused this mischief. I have from time to time reported these facts to breeders who have called upon or written me in relation to abortion in their herds, and several have used care in regard to hay seeds with good results.

I notice that the veterinarians employed by the Government to investigate the outbreak of alleged foot-and-mouth disease in the West state that the malady is not the contagious disease it was supposed to be, but was caused by ergot in the hay. Now, if ergot in the hay will "contract the blood-vessels and retard circulation," as reported by these surgeons, have we not discovered the cause of abortion in cows that eat such hay? In my case I feel quite sure that I have discovered a cure, viz, feed with hay free from ergot. I do not think hay seed from the West should be sold here until after it is thoroughly inspected.

TUBERCULOSIS.—Dr. J. A. Rice, of Liberty Mills, Orange County, Virginia, in January last reported the following cases to the Department:

Last summer a young steer in our herd that had been improving so rapidly as to be the subject of remark was one day found apparently suffering with rheumatism. He continued to decline until sold. I afterwards learned that on being slaughtered his lungs were found very much diseased. In October two of our dairy cows (one of them very fat) were taken with symptoms very similar to those of the steer—separating themselves from the herd, stiff in the shoulders and neck, loss of appetite, costiveness, rapid shrinking in their milk, no cough, and no symptoms by which we could diagnose the disease. The decline has continued without any special symptoms except those mentioned, aside from a great shrinkage in flesh.

Yesterday we slaughtered one of the cows to see if we could not find the cause of the disease. We found the heart and lungs very large and filled with tubercles. The omentum felt like a hard, round cord, and was filled with tubercles, in fact seemed to be one tuberculous mass. We found the liver, spleen, stomach, and intestines all healthy. In cutting into the tubercles they presented more of a sarcomatous appearance than of true tuberculosis. Thinking the specimens might be of interest to your veterinarian, I have boxed up the lungs and omentum and forwarded to your Department, and would be glad to hear the result of the examination. These cattle were all bred upon the farm. I have bred their ancestors on dam side for more than thirty years. They were superior dairy cattle, of fine constitutions and remarkably healthy. I never knew one of them to be sick before. They were of different strains—the sire of the cows was a Mazurka bull by Royal Oxford, of the steer a Miss Kirby cow by the Rhenish bull Joe Johnston. If this is true tuberculosis, how did it originate?

A day or two later (February 1) Dr. Rice writes as follows concerning the second cow:

We killed the other cow to-day, and found such a growth of fungoid matter that I hasten to send specimens, believing they will aid your veterinarian to form a more correct diagnosis of the cases. From all I can learn about the steer, he presented similar growths. They are remarkable, and owing to the previous healthfulness of the animals, and that of their ancestors on all sides, I am able to form no opinion in the premises. You will find myriads of small fungi growing from the larger ones—some much resembling mushroom growths. The lung presented very much the condition of the one we forwarded yesterday. We to-day send liver entire, its preternatural adhesions to midriff and pectoral coat of stomach by fungous growth; a section of rib and brisket, showing growth of fungi to the inside membrane of the ribs; sections of lung, peritoneal covering of bowels, omentum, &c.; also a piece of stomach with growth growing from its outside coat. All of the interior of the ribs were covered with growths such as specimens represent, and many with dark bodies hanging attached by long, narrow necks to the membrane between the ribs, resembling hydatids, but the bodies were hard and fleshy.

There has never been any cough with either animal, only a gradual decline with the symptoms given in my first letter, except that the one we killed to-day was taken a day or two ago with a profuse, watery diarrhea, which was weakening her down rapidly.

The specimens arrived in good condition, and, after examination by the veterinarian, Dr. Rice was informed that the disease with which his cattle were suffering was undoubtedly tuberculosis. He was counseled to destroy all remaining animals showing signs of the malady, and advised to thoroughly disinfect his stables, which should not be again occupied for a year.

TRANSMISSION OF TUBERCULOSIS.—Mr. Charles Pidgeon, of Wadesville, Clarke County, Virginia, wrote as follows to the Department under date of September 15, last:

We have a lung disease among our cattle which we think is contagious pleuro-pneumonia. It is pronounced to be such by our resident physician and by cattle men who have had experience with the disease. We have been treating the animals all summer without any good effect, and would now like advice as to what further to do to get rid of it. We have lost two cows, and have a number of others sick. We examined the cows after death and found their lungs very much swollen, covered with tubercles, and growing fast to the ribs.

Dr. W. H. Rose, of the veterinary experimental station, was at once directed to visit and inspect this herd for the purpose of determining the nature of the disease. This he did on the 16th of September. Mr. Pidgeon's herd consisted of seventeen Devons, of both sexes, the majority of them being young animals. The history of the animals and the symptoms indicated tuberculosis. Dr. Rose condemned three of the milk cows, one of which he slaughtered for *post-mortem* examination. The autopsy revealed extensive deposits of tubercles in both lungs. He advised the slaughter of the other condemned animals as a precaution against the further spread of the disease.

The disease in this herd originated from a bull purchased some four years ago by Mr. Pidgeon in the vicinity of Baltimore, Md. This bull died of a lung disease about one year after its arrival on this farm. Since then several cows and some of this bull's calves have died of the malady.

The Department was recently advised by Mr. Q. C. Babcock, residing near Fayette, Iowa, that his cattle were suffering with disease, and requested that a veterinarian be sent to make an examination of the affected animals. Dr. M. R. Trumbower was directed to visit the herd and give such advice as the nature of the malady might require. Under date of December 9, 1884, he writes as follows concerning his visit to this herd:

I this morning drove out to the farm of Mr. Q. C. Babcock, a distance of eight miles from this place, and examined his cattle, viz., 2 middle-aged cows, 1 of which is affected with tuberculosis, and the other some bronchial affection, probably of entozoon origin. Both have some cough, especially in the morning.

Four calves out of 92 head died with symtomatic anthrax during the summer. The last death occurred about two weeks ago. One calf died about the same time which had been affected with a cough for several months, and on post-mortem examination revealed a number of *strongyli* in the trachea. About two-thirds of his calves have more or less of a cough, but otherwise are in good order, with the exception of about 6 head. These are rather thin in flesh, and the cough is harassing. All the conditions present would indicate parasitic bronchitis. He has been administering sulphurous anhydride, ferri sulphas in drinking water, and ginger and gentian in the feed.

IMPACTION OF THE MANIFOLD.—Mr. P. H. Powers, of Wickliffe, Clarke County, Virginia, statistical correspondent, recently lost some cattle by a disease unknown in that locality. Under date of February 18 last, he wrote the Department as follows:

I have lost, within the past ten days, two three-year-old steers, affected similarly and so acutely that I deem it proper to report the cases to the Department. When first observed, which was about 6 o'clock a. m., each of these steers seemed suffering with violent itching about the head, particularly along the jaw and around the eyes. So violent was this that they would run to the fence or posts of the barn and rub until the whole side of the head was made raw and bloody. Accompanying this itching was a spasmodic twitching or jerking of the head sideways and upward, occurring constantly, and at intervals of about a minute. There was some foam about the mouth. So acute was the disease that each animal was dead in less than ten hours. No other symptoms were observed. These steers were in good condition. One was raised on the farm, and the other bought in Chicago in November last.

Later, on February 25, Mr. Powers again wrote the Department giving additional information in regard to the progress of this disease. He says:

I have since lost another very fine animal with symptoms identical with the others. Death ensued within twenty-four hours. These cattle were all in fine condition, have

> been well cared for and had access to water, and were fed on corn-fodder and wheat-straw. In fact, the management has been the same that I have given my stock cattle in the winter months for thirty years. The difficulty seems to be that no evidence of disease or ailment appears which is noticeable until the animal is in *extremis mortis*, when all remedies would seem unavailing. A farmer living some ten miles from me in this county has lost, I understand, some seventeen head this winter. As soon as I can ascertain the facts I will report them.

On receipt of Mr. Powers' first letter, he was informed by the Chief Veterinarian of the Bureau that his cattle were suffering with impaction or obstruction of the manifolds from eating too much dry food, and advised to give full doses of purgatives combined with stimulants.

The following deaths described by Mr. J. B. Warren, statistical correspondent of Rooks County, Kansas, no doubt occurred from acute indigestion or impaction:

> One of my neighbors has lost six head of cattle within the past ten days. They died within from one to three hours after the first symptoms were observed. I opened four of them, but found nothing wrong with any of the organs except the stomach and bowels. The inside coatings of these seemed as though badly eaten with lye or some other strong substance. There was very little blood, and around the stomach it was settled in black clots. A foamy froth, resembling thick, slimy soap-suds, passed from the animal's mouth while suffering from the disease. Before death they bloat badly. All the animals attacked were in apparent good health and excellent condition up to the time the first symptoms were observed.

CATTLE POISONED BY VEGETATION.—Mr. George W. Carleton, Gayoso, Pemiscot County, Missouri, writes under recent date as follows:

> Since the February overflow of the Mississippi River, a great many cattle have died, especially cows. They are affected with weakness in the loins, break down, drag their hind legs, fall down and cannot rise, thrash their heads upon the ground, and die within two hours after being attacked. I assisted in the dissection of a two-year-old heifer that died within an hour after showing symptoms of the malady. Upon opening the stomach we found a quantity of an undigested root of a vine that grows in great abundance here, known in the country as "cow itch," trumpet-flower, or cow-vine—*Bignonia radicans*. Near the bank of the Mississippi River, where the current in the "back-water" runs very strong, the soil has been washed off and the roots of this vine left exposed, and, being very tender, cattle eat them ravenously. All the inner coating of the stomach was of a very dark purple color; on the spleen were found a few parasitic worms; about two inches square of the spleen was inflamed and appeared to have been perforated. In all probability these parasites had caused the damage. Upon removing the skull we found a great pressure of blood, serum, and water on the brain. This was no doubt caused by the action of the poisonous roots found in the stomach, and was the immediate cause of the animals' death. Within the last ten days several farmers have lost valuable cows, and all of them have eaten of the root of the *Bignonia radicans*."

Mr. J. G. Knapp, Limona, Fla., who is the statistical agent of the Department for that State, forwards the following extract from a letter addressed to him by Mr. F. A. Hendsy, of Fort Myers (Caloosahatchee), Florida, in April last:

> Some cattle have dropped dead in this locality, number not known. For several years cattle have died here from this unknown cause. So far the cause is a mystery. A few years ago one thousand or more animals died in fat condition. All ages and sexes were alike affected. There is no lingering; apparently as sound as can be and in a minute of time lifeless. It is not contagious—prevails only in certain localities. I have dissected the animals and examined carefully, but found no internal evidence of disorganization.

Mr. Knapp adds that the indications are that some very active vegetable poison is the cause. Several very poisonous plants and trees, such as the *Hippomane* and other *Euphorbiaceæ*, with the *Rhus metopium*, grow there.

HORSES.

HORSES POISONED BY VEGETATION.—A correspondent at San Antonio, Tex., informs the Department that there have been heavy losses of horses through a strip of country where the live oak, red and post oak, and the hickory grow. This district runs through Medina, Atascosa, Bexar, McMullen, Live Oak, Wilson, and Frio counties, and ends about the boundary line of De Witt. The land in this district is generally of a sandy nature, and does not produce a vigorous growth of any variety of grass, though it is for the greater part of the year, on an average, a fair range country. At this time of year (April) there is little grass in this district, and stock have been subsisting partially, for some time, on acorns, to the eating of which the losses of horses are attributed. Mr. Duck, Mrs. O'Brien, and others, have lost quite a number of animals, and it is reported there have been some losses in W. W. Robbins' pasture, where stock has been placed for fattening. The horses, when first attacked, seem to be drawn together as if in loin distemper, and appear to suffer greatly, virtually walking on the tips of their hind hoofs. Then they gradually pitch forward, fall to the ground, and never get up again. The affliction does not seem to have any relation to the condition of the animal, as some of the fattest horses in the country have fallen a prey to the malady. Mares seem to suffer most. Mr. Duck attributes the losses to acorns, which are not only wormy, but were not so well matured as in former years. Hogs, however, have grown very fat on them.

TERRIBLE DESTRUCTION OF FARM ANIMALS BY BUFFALO GNATS.—The losses of farm animals last spring in some of the Southern States from the irritation and poison of buffalo gnats were very heavy. No deadly contagious disease has ever caused such destruction among the herds and flocks of the Southwest as have these poisonous pests during the past season. Mr. A. F. Osborn, Winnsborough, Franklin Parish, Louisiana, writing to the Department under date of May 15 last, says:

> Fully one-half of all the farm animals of this parish have fallen victims to the poison impregnated by the bite of the buffalo gnats. Horses, mules, cattle, sheep, and hogs have alike succumbed to the scourge, and there seems to be no abatement except in localities where the material to act upon has disappeared or been exhausted. Some persons have lost all, others two-thirds, and he is indeed fortunate who has saved half his stock. The dead carcasses lie bloating around fields and on highways, and nothing—not even a buzzard—will touch them. The stench arising from these putrid carcasses is almost intolerable, and fears are entertained that a pestilence will follow.
>
> On the 22d of April we collected partial statistics from two of the nine wards into which the parish is divided. As far as we were able to ascertain the deaths in those two wards amounted, at that date, to 3,187. Many individuals, in handling and skinning the animals, have been poisoned. Some have already died, others will probably die, while still others will escape with the loss of a hand or an arm.

Mr. Thomas B. Gilbert writes from Oakley, La., under date of June 29 last, as follows:

> About the middle of April last a fatal disease broke out among cattle, horses, mules, hogs, and sheep in this parish (Franklin) and the adjoining parish of Richland. It attacked all of the above-named animals almost simultaneously, making its appearance first among the cattle a few days after the dreaded buffalo gnats came in. The time for the buffalo gnats to make their appearance here varies with the heat or cold of winter. They came earlier in 1882 than I ever knew them to come before—say, about the 5th of March; but the usual time is from the 1st to the 10th of April. This year they came about the 1st of April, and in a few days multiplied into millions, spreading over the entire country, and no animal could survive their attacks many hours unless protected by smoke. They have a great aversion to smoke, and this is the only protection our animals have from their ravages. Work animals are greased as an additional protection. The gnats were more numerous this year than common, but not more so than they were in 1882. In this parish (a small one) about 3,000 head of horned cattle died in a few days, and about 300 head of horses and mules, 5,000

or 6,000 head of hogs, and as many sheep. The horses and mules are still dying at intervals. In these the disease assumed the form of charbon; it did the same with many cattle; and what is singular. the only cattle, horses, and mules that recovered were those that it attacked in that form. All those attacked in the other form of the disease (*i. e.*, without external swelling) died. Now, what could have occasioned this dreadful and fatal plague among our domestic animals? The disease appeared in a belt of country, say 25 miles in extent from north to south, and extended at least that distance east and west. North of that line there was no disease, and south of it there was none. How far west on that parallel the disease extended I do not know. But on the east the Bayou Macon was the line of demarcation.

It is safe to say that the people of Franklin and Richland parishes have lost more than $150,000 worth of cattle, horses, mules, sheep, and hogs from this fatal disease. I think that next year a competent man from your Department ought to be sent into every county of Arkansas and Mississippi, and into every parish in Louisiana infested with buffalo gnats to study their habits, origin, and the cause of the disastrous effect they are yearly having upon the inhabitants and the domestic animals of the infested district. This pest is assuming such alarming proportions as to threaten the depopulation and abandonment of all the high-land country adjacent to or bordering upon the overflowed regions of the Mississippi Valley. It is not confined to the valley proper, but the highlands contiguous to, and for many miles inland, are worse cursed with the buffalo gnats than the lowlands themselves. I was born and raised here, am 46 years old, and never saw or heard of the pests until 1865. They have come with the annual overflows every year since, and their ravages are so far extended over this region of country, and so fatal to man and beast, that the continued occupation of the country is problematical. Some deaths and many cases of charbon have occurred among our people, both black and white.

DISEASE AMONG HORSES IN FLORIDA.—Mr. S. Stringer, statistical correspondent for Hernando County, Florida, writing under date of December 29, 1884, gives the following description of a disease which is afflicting horses in that locality:

It makes its appearance near the feet or mouth, and sometimes on the side about the flank, and, in fact, any part of the body is liable to it when exposed to the cause, whatever that may be.

The first symptom observed is an oozing of bloody serum or watery fluid from an abraded surface, with a hardened, lobulated feeling. If, during this state, it is immediately excised and cauterized, and the animal taken off the pasture and stabled, it will heal, but if not discovered it will burrow deep in and around the fetlock or leg, or any other locality, and will even penetrate joints, and sometimes will perforate the wall of the abdomen and of course terminate fatally. I have known it to literally fill up the nares and so honeycomb the palate as to destroy life in a few months. The fleshy parts around the diseased portion, or the parasite, if it is one, become very hard, and has the appearance when cut of ligamentous tissue. This, I presume, is organized lymph thrown out in consequence of local inflammation. It is sometimes striated, and appears very much like schirrus matter.

Horses get this disease in heat of summer while grazing on meadows, on what are known in Florida as prairies. It is more apt to occur during rainy seasons. Some horses do not seem to be susceptible, while others will take it by one day's grazing on the prairie.

In the sinuses or tortuous cavities of this terrible disease you will discover hard, horny, and irregularly-shaped lumps of curdy or cheesy matter. The disease is known by the people of this county as "leeches," and the hard, cheesy lumps referred to are called the "leeches." This, of course, is a misnomer, as the lumps seem to be animal tissue hardened, resembling tuberculous matter, not yet softened. The specimen sent is a very fair sample, and was taken this morning from a fine horse of mine near his hinder fetlock. He has suffered since last July, and was operated on two months ago unsuccessfully, and doubtless will be lost in conseqence of the joint becoming implicated.

VARIOUS REMEDIES AND PREVENTIVES.—Mr. Byron L. Saunders, Purdy, Tenn., writes as follows, under date of April 5, last:

The new disease which has recently made its appearance among cattle in Illinois and Iowa prevailed among my cattle last winter a year ago. If taken in time it is very easy to manage. Strong apple vinegar, or acetic acid, and blood-root—a strong decoction or tincture, applied three times a day will cure it.

For swine plague and fowl cholera: Equal parts of soda, alum, and copperas, and one-tenth part of blue vitriol. To prevent the plague, feed mandrake to the animals in their slop.

For murrain in cattle, Spanish fever, and inflammation of the liver: One-half cup of lobelia seed, 1½ cup of blood-root, ½ cup mandrake, 1 cup wild-cherry bark, 2 cups dog-fennel blossoms, a piece of garget, or poke-root, the size of a small hen's egg, to which add one gallon of water and boil down to one pint and a half of solution. This is a dose for a grown animal, and if given in time will generally effect a cure in the above-named diseases.

Mr. L. A. Cook, Milledgeville, Ga., states that twenty-five drops of tincture of aconite given when symptoms are first observed is almost an infallible remedy in any form of colic. He has never known it to fail, and says that a second dose is rarely necessary. He regards it as the simplest and surest of all remedies.

HOGS.

BREEDS AND SANITARY CONDITION.—Mr. Henry C. Mosely, Lawrence, Kans., regards our present breed of hogs and their sanitary condition all that could be desired. He writes to the Department as follows:

I have traveled for fifteen years in the great hog-producing regions of the Mississippi Valley, and have therefore no hesitancy in declaring that the sanitary condition of swine is now better than at any period in the past twenty years. And why? Because the swine producers are provided with acclimatized and better breeding animals, and are not now introducing new stock, which the farmers all admit has been one of the most prolific causes of disease. The most extensive swine producers now allow their animals to range in pastures during the summer months; feed them less corn; provide more and better water; are watching them more closely, and gradually reducing the rearing of this class of animals to a science. The American hog is now, in my humble opinion, all right, and the way to continue its prosperity is for the non-swine producers to adopt the non-intervention policy, or hands off, and leave it where it now is, at the front.

EFFECTS OF FEEDING SWEET POTATOES.—Mr. J. G. Knapp, statistical correspondent for the State of Florida, writes as follows under date of April 10 last:

Allow me to call your attention to a remark made by J. M. Strickland, my correspondent from Putnam County, which is new to me. He says:

"It is thought that feeding potatoes (to hogs) during winter is the cause of cholera. Last year I lost all the hogs that I put on my potato ground, and this winter they came off in poor condition, with a loss of 25 per cent."

Potatoes here mean the sweet potato, *Batata edulis*. The hogs are placed in the grounds to root for those that are left after the crop is dug. These potatoes remain sound in the ground during the winter, as there is not frost sufficient in many portions of the State to destroy them if disconnected with the vines. Can there be anything in these potatoes to produce cholera?

Cholera or swine plague is a purely contagious disease, and can only be communicated to the animal by coming in contact with the virus. Possibly sweet potatoes might render hogs more susceptible to the disease than some other kind of diet.

SWINE PLAGUE IN ARKANSAS.—Mr. Felix G. Davis, of Grand Lake, Chicot County, Ark., writes as follows under date of March 5:

Through the kindness of Senator Garland and Hon. P. Dunn, of this State, I have received three copies of the Report of the Department of Agriculture for 1883, two of which I distributed to my neighbors. I think a copy ought to be in the hands of every intelligent farmer. The reports on swine plague, chicken cholera, and charbon are of great interest to us, especially the report on swine plague, or cholera, as it is vulgarly termed. On this Isle of the Swamps, or Mason Hills, as it is called, we are now being visited by this hog disease to an alarming extent. Fine stock hogs that in January were fat and healthy are now dying on every farm, and those left are generally poor in flesh. Being deprived of their usual range by an overflow of the swamps has no doubt had much to do with the spread of the disease.

SHEEP.

NEW DISEASE AMONG LAMB.—Some months ago Mr. G. W. Marshall, Eckley, Carroll County, Ohio, wrote as follows concerning a disease which at that time was destroying a great many spring lambs in that locality:

> We have a strange disease among our spring lambs here this spring that I think should be investigated by the Department. They die when they are from three to five weeks old. Apparently the very best lambs in the flocks will be well, as far as you can see, in the morning, and by night will be dead. They act as though they had spasms or fits. We have lost six, and some farmers have lost as high as thirty or forty this year. It is not in all flocks, just here and there. Sometimes you will find five or six lying dead at a time. I hear of it in places all over the county. We cannot tell what the disease is, nor have heard of any remedy for it. Some claim they get too much milk; others say that is not the trouble. Some call it lamb cholera.

In answer to a letter of inquiry asking for further information as to the symptoms of the disease and the *post mortem* appearances of the animals that had died of it, Mr. Marshall writes:

> No more lambs have died since receiving your note, hence I have had no chance to make a *post mortem* examination. However, a neighbor says he examined several. and there appeared to be water about the heart, an unusual amount; the gall appeared dark and slender, as though rolled up; stomach and other parts all right, as far as he knew. The lamb when first observed seems entirely helpless. It then has spells as though affected with a fit; plunges about, works its ears and mouth, rolls its eyes and froths at the mouth. The animals generally attacked are from three to six weeks old, and are usually those in the best condition. They live from six to twelve hours after the first symptoms of the disease are observed. We took our ewes off good blue-grass, white clover, and timothy pasture and put them in a woods pasture, after which we lost no more lambs. My observation has been that the lambs that have died have been those that have had the greatest amount of milk from their mothers.

A FATAL LOCAL DISEASE.—A fatal disease, which is no doubt local in its character, is described as follows by Mr. L. F. Dupron, living near Savannah, Ga.:

> A disease is prevailing here among sheep which seems to be peculiar to animals feeding on the seaboard. The principal symptom is a swelling on the under jaw. The swelling contains a hot, watery fluid; if opened and the fluid discharged it will soon fill again with increased heat over the affected part. Diarrhea sets in, which is soon followed by death. Sheep over three years old frequently die before diarrhea occurs. The disease is most fatal to lambs, though I have known but two recoveries out of hundreds of animals attacked. Sheep grazed in woods pastures seem as liable to the disease as those grazed in the salt marshes.

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